Journal of
CHRONIC
DISEASES

VOLUME 12 . NUMBER 6

DECEMBER, 1960

OL.
12
960

Editorial

THE DAILY DOSE OF FOLIC ACID

WILLIAM H. CROSBY, COLONEL, MC, USA
WASHINGTON, D. C.

From the Department of Hematology, Walter Reed Army Institute of Research, Walter Reed Army Medical Center

(Received for publication July 30, 1960)

ON July 14, 1960, the Food and Drug Administration announced its intention to issue a statement of policy concerning the content of folic acid in multivitamin preparations[1]: "Although not itself harmful, folic acid, when added in sufficient dosage to the diets of persons suffering from pernicious anemia who are not receiving proper medication, has the capacity of correcting the blood disorders of this disease while the accompanying nervous system changes may progress. This may create a hazard for a very few individuals who are undiagnosed, untreated victims of pernicious anemia. This situation is not completely corrected by the incorporation of vitamin B_{12} and intrinsic factor in folic acid-containing preparations for oral use." The Food and Drug Administration's regulation is intended to prevent the incautious administration of folic acid to patients with unrecognized pernicious anemia and requires that vitamin preparations be sold only by prescription when they contain more than 400 μg (0.4 mg.) of folic acid per daily dose. For some years this problem has been a subject of debate between hematologists (who regard each of these crippled patients as the victim of an avoidable tragedy) and nutritionists (who tend to regard humanity in terms of mass and folic acid as a food). The hematologists, and others, may wonder why the Food and Drug Administration stops where it does. If the intent is to protect these patients, why is it safe to permit 400 μg of folic acid?

The Food and Drug Administration's decision was based upon a statement published by the Council on Foods and Nutrition of the American Medical Association[2]: "Recommended Dietary Allowances have not been established for pyridoxine, pantothenic acid, folic acid, or vitamin B_{12}, although all these substances are essential in human metabolism. The amounts present in diets con-

sidered adequate in other factors should serve as a guide for quantities to be used in supplementation. The amount of folic acid in supplementary vitamin mixtures should be no greater than that available from an abundant dietary. Common experience indicates that this is a quantity which will seldom support hematological function in pernicious anemia and, therefore, will not mask the diagnosis of this disorder. Although this quantity is still to be determined, 0.3 mg. of folic acid is suggested tentatively as a proper maximum amount for supplemental mixtures."

Since January, 1959, when the Council's statement appeared, additional information has developed which bears upon this point.

The work of Sheehy and associates[3,4] relates to the portion of the Council's statement concerning the amount of folic acid available from an abundant diet. The values which have been generally accepted as representing the concentration of folic acid in foods were established by means of bioassay, according to the ability of various food substances to support the growth of folic acid-requiring organisms.[5] These values are questionable, since it has been shown that the requirement of the assay organisms is not strictly for folic acid (pteroylglutamic acid).[6] It was possible to use fragments of folic acid and other glutamates in place of pteroylglutamic acid. Hence, the results of bioassay are high when interpreted in terms of pteroylglutamic acid. The error would be of no importance to problems of human nutrition if the human organisms were as versatile as the bacterial.

However, Sheehy and his collaborators,[3,4] working in the U. S. Army Tropical Research Medical Laboratory in Puerto Rico, have found that humans evidently have a specific requirement for pteroylglutamic acid or closely related molecules. They have demonstrated that the megaloblastic anemia of tropical sprue can be corrected by as little as 25 μg per day of pharmaceutic folic acid administered by mouth. The bone marrow quickly reverts to a normoblastic pattern and the hemoglobin level begins to increase, sometimes without any spike of reticulocytes. The remission occurs while the patients are maintained on the same diet they received while they developed the disease, a diet which contains, as determined by bioassay, 500 to 1,500 μg of folic acid. Analysis of the patients' intestinal juice demonstrates normal conjugase activity, so, presumably, they are able to split the conjugated folic acid in food. This supplementation by 20 μg of pharmaceutic folic acid represents about a 5 per cent increase of folic acid available to the patients, if it is assumed that the dietary folic acid was pteroylglutamic acid or similar molecules which could satisfy the human requirement. The assumption does not seem justified. The human organism does not seem capable of substituting the fragments for the folic acid.

The bioassay performed on the patient with sprue seems to indicate two things: (1) that the concentration in food of folic acid useful to humans is lower than previously surmised and (2) that the folic acid requirement in the human is correspondingly lower.

With the perspective provided by the work of Sheehy, it would not be surprising to learn that the daily requirement in normal humans is of the order of 5 μg of folic acid.

In this perspective, a supplemental vitamin preparation containing 400 μg of folic acid would provide many times more than the daily dietary intake.

A second group of reports is concerned with the response to folic acid of patients with pernicious anemia. Marshall and Jandl[7] studied 3 patients with megaloblastic anemia of pernicious anemia. After a brief control period when the patients received no treatment, each patient was given intramuscular injections of 400 μg of folic acid per day for a period of 10 days. The hemoglobin and reticulocyte concentrations of the blood were followed and demonstrated "little or no response." On the basis of this experience, the authors conclude: "From 200 to 500 micrograms of folic acid daily orally should suffice for the prevention of dietary deficiency of folic acid without endangering patients with undiagnosed pernicious anemia." One wonders if the data merit such confidence, questioning not the results but the conditions of the experiment. An experimental situation lasting 10 days is not analogous to that of a patient with unrecognized pernicious anemia who unwittingly receives folic acid in a multivitamin preparation for many weeks or months. A case reported recently by Ellison[8] illustrates the point. The patient had mild anemia (10 to 12 Gm. hemoglobin). Over a period of 4 months, she took a twenty-two ingredient vitamin preparation which contained 330 μg of folic acid per capsule. She had taken one or more per day, believing "If one is good, two or three should be better." When she appeared with symptoms of combined system disease of 2 months' duration, she had no anemia (14 Gm. hemoglobin), and the bone marrow was normal. The sensory and locomotor defects were only partially corrected by injections of vitamin B_{12}.

Such a case demonstrates a possibility that "small" doses of pharmaceutic folic acid can correct pernicious anemia if taken over an extended time. The Food and Drug Administration's memorandum[1] points out in addition, "We do not know to what extent the addition of 400 micrograms of folic acid per day may *delay* the onset of anemia in patients who develop true pernicious anemia and, therefore, increase the chance of neurological change."

Until the matter has been clarified, it seems wise to reduce to a minimum the risk to these patients by permitting *no* folic acid to be sold without prescription.

REFERENCES

1. Larrick, G. P.: Status of Folic Acid in Foods for Special Dietary Use and as a Drug, Federal Register **25**:6633, 1960.
2. Council on Foods and Nutrition, American Medical Association: Vitamin Preparations as Dietary Supplements and as Therapeutic Agents, J.A.M.A. **169**:41, 1959.
3. Perez-Santiago, E., Sheehy, T. W., and Rubini, M. E.: Primary Folic Acid Deficiency in Tropical Sprue, Clin. Res. **8**:215, 1960 (Abst.)
4. Sheehy, T. W.: Personal communication.
5. Toepfer, E. W., Zook, E. G., Orr, M. L., and Richardson, L. R.: Folic Acid Content of Foods, Microbiological Assay by Standardized Methods and Compilation of Data From Literature, U. S. Department of Agriculture Handbook 29, Washington, D. C., 1951, U. S. Government Printing Office.
6. Jukes, T. H., and Stokstad, E. L. R.: Pteroylglutamic Acid and Related Compounds, Physiol. Rev. **28**:51, 1948.
7. Marshall, R. A., and Jandl, J. H.: Responses to "Physiologic" Doses of Folic Acid in the Megaloblastic Anemias, A.M.A. Arch. Int. Med. **105**:352, 1960.
8. Ellison, A. B. C.: Pernicious Anemia Masked by Multivitamins Containing Folic Acid, J.A.M.A. **173**:240, 1960.

RELATIONS BETWEEN HARDNESS OF WATER AND DEATH RATES FROM CERTAIN CHRONIC AND DEGENERATIVE DISEASES IN THE UNITED STATES

HENRY A. SCHROEDER, M.D.
WEST BRATTLEBORO, VT.

From the Department of Physiology, Dartmouth Medical School and the Brattleboro Retreat

(Received for publication April 9, 1960)

VOL. 12 1960

DEATH rates vary widely from state to state. Significant negative correlations between weighted average hardness of water supplies and death rates from cardiovascular diseases in the United States have been reported.[1] In States supplied with harder-than-average water, average annual age-adjusted death rates from all causes and from cardiovascular diseases for both sexes, for males, for females, and for white males aged 45 to 64 years, were in general lower than national averages. These relationships applied to death rates from coronary heart disease as well as to rates from all other cardiovascular diseases. Rates for coronary heart disease in older white males correlated significantly with hardness and with a number of constitutents of drinking water in the 163 largest municipalities of the country. Correlations were absent for deaths from all other causes.[1]

In order to define these interesting relations further, the present report examines correlations by state between weighted average hardness of "finished"* water (an index of exposure per person) and average annual death rates from a number of chronic and degenerative diseases for the years 1949 to 1951.[2] Correlation coefficients (r values) were calculated by the usual method.[3] Values for water were measured in 1950 to 1951.[4] Diseases listed among the ten leading causes of death and subdivisions thereof with relatively high death rates in the decade 55 to 64 years were chosen in order to avoid, as much as possible, rates possibly erroneous because of small numbers of cases. This consideration applied in general to death rates less than 10 per 100,000 persons.

States which have harder water than the national average are all those in the East North Central (5 states), West North Central (7 states), and Mountain

This work was supported by a grant in aid from Ciba Pharmaceutical Products, Summit, N. J., and by a grant from the U. S. Public Health Service, Grant H-5076.

*"Finished" water is water which has been treated in municipal treatment plants by procedures such as chlorination, filtration, softening, adjustment of pH, control of algae, removal of iron and manganese, coagulation, and aeration. As such, it is the water delivered to the tap. "Raw" water has received no treatment.

(8 states) areas, plus Florida, Kentucky, Oklahoma, Texas, and California.[5] Therefore, 25 states have water harder and 23 states and the District of Columbia have water softer than the national average of 97 parts per million.

It so happens that many "soft-water" states have large nonwhite populations. The correlation coefficient of hardness of water and per cent of the nonwhite population per state was −0.341, which is significant at the 2 per cent level of confidence. Therefore, death rates of white persons were analyzed in order to avoid this influence. In all cases, however, correlation coefficients were calculated as well for all races and, in most cases, for all ages; these data are reported when they appear of interest to the problem.

RESULTS

In Table I are shown the correlation coefficients and their levels of significance for water hardness by state and death rates from the ten leading causes of death for all ages, races, and both sexes. Significant values appear mainly in the cardiovascular group of diseases, motor vehicle accidents, and congenital malformations. In Table II, confined to white persons aged 55 to 64 years, major categories are subdivided into their constituent parts. In white persons the

TABLE I. CORRELATIONS BY STATE OF WEIGHTED AVERAGE HARDNESS OF WATER AND AGE-ADJUSTED DEATH RATES FOR THE TEN LEADING CAUSES OF DEATH, 1949 TO 1951 (PER 100,000 POPULATION; ALL AGES, RACES, BOTH SEXES)

CAUSE OF DEATH*	UNITED STATES RATE	r	p
All causes	844.4	−0.36	0.01
1. Cardiovascular (330-334, 400-468)	423.4	−0.56	<0.01
2. Malignant neoplasms (140-205)	125.4	−0.309	<0.05
3. Vascular lesions affecting central nervous system (330-334)	88.6	−0.334	0.02
4. Accidents			
Motor vehicle (E 810-835)	23.1	+0.463	<0.01
(white only)	22.9	+0.457	<0.01
Other accidents (E 800-802, E 840-962)	35.0	+0.252	NS† (0.1)
(white only)	33.5	+0.306	<0.05
5. Certain diseases of early infancy (1957 rates)‡ (760-776)	39.1	+0.145	NS
Infant deaths under 1 year per 1,000 live births‡	23.3	+0.224	NS
6. Influenza and pneumonia (480-493)	25.9	−0.118	NS
Same (1957 rates)	35.8	−0.064	NS
Pneumonia (490-493)	22.5	−0.074	NS
Pneumonia (under 1 year)	249.9	+0.132	NS
7. General arteriosclerosis (450)	16.4	−0.164	NS
8. Diabetes mellitus (260)	14.5	−0.178	NS
9. Congenital malformations (750-759)	12.4	+0.494	<0.01
Same (1957 rates)	12.8	+0.299	<0.05
Same (under 1 year)	458.6	+0.289	0.05
10. Cirrhosis of liver (581)	8.8	−0.218	NS
Symptoms, senility and ill-defined conditions (780-795)	15.2	+0.079	NS

Data from References 2, 4, 5.

*Numbers in parentheses refer to International List of Diseases and Causes of Death, Sixth Revision, 1948.

†N.S. = Not significant.

‡Not available in Reference 2.

highest negative correlations occurred with those disorders commonly believed to be associated with atherosclerosis and with arterial hypertension, (hypertension with heart disease, vascular lesions affecting the central nervous system, general arteriosclerosis, and arteriosclerotic heart disease, including coronary disease); correlations were not significant in the case of rheumatic heart disease, nonrheumatic chronic endocarditis and other myocardial degeneration, chronic nephritis, or "other diseases of heart" (Table II).

In the case of malignant neoplasms, one probably significant negative correlation ($p = 0.02$) appeared for neoplasms of the respiratory tract in males aged 55 to 64 years. In this decade there was some relationship ($p < 0.05$) between water hardness and death rates from all neoplastic diseases and from those of unspecified sites; for other carcinomas, lymphomas, and leukemias, there were no correlations of significance.

VOL. 12 1960

TABLE II. CORRELATION BY STATE OF WEIGHTED AVERAGE HARDNESS OF WATER AND AVERAGE ANNUAL DEATH RATES FOR MAJOR DISEASES, 1949 TO 1951, WHITE PERSONS AGED 55 TO 64 (PER 100,000 POPULATION)

CAUSE OF DEATH	CATEGORY*	UNITED STATES RATE	r	p
Cardiovascular diseases				
Arteriosclerotic heart disease				
Male	420	811.6	−0.498	<0.01
Female		266.7	−0.455	<0.01
Vascular lesions affecting central nervous system	330–334	169.5	−0.34	<0.02
Hypertension with heart disease	440–443	99.9	−0.567	<0.01
Hypertension without mention of heart	444–447	11.8	−0.289	<0.05
General arteriosclerosis	450	10.5	−0.335	<0.02
Chronic endocarditis, etc.	421–422	68.8	+0.004	NS†
Chronic rheumatic heart disease	410–416	31.7	+0.103	NS
Other diseases of heart	430–434	29.4	+0.138	NS
Chronic nephritis, etc.	592–594	25.8	−0.186	NS
Noncardiovascular diseases (all ages, races)		420.5	+0.02	NS
Total white male, aged 45-64		718.2	−0.10	NS
Malignant neoplasms	140–205	385.9	−0.317	<0.05
Of digestive organs	150–159	147.5	−0.205	NS
Of male respiratory system	160–164	95.4	−0.332	0.02
Of female genital organs	171–179	82.4	−0.263	NS
Of female breast	170	71.4	−0.084	NS
Of other and unspecified sites	156B–165 190–199	38.6	−0.294	<0.05
Lymphosarcoma, etc.	200–203, 205	14.9	−0.149	NS
Leukemia and aleukemia	204	12.5	+0.052	NS
Diabetes mellitus	260	41.7	−0.227	NS
Cirrhosis of liver	581	28.6	−0.324	<0.05
Ulcer of stomach and duodenum	540–541	16.7	+0.006	NS
Pneumonia	490–493	28.8	−0.119	NS
Congenital malformations				
All ages	750–759	12.6	+0.464	<0.01
Less than 1 year of age		477.4	+0.165	NS
Symptoms, senility and ill-defined conditions	780–795	12.4	+0.245	NS (0.1)
Hernia and intestinal obstruction	560–561, 570	10.6	+0.247	NS (0.1)

Data from References 2, 4.
*International List of Diseases and Causes of Death, Sixth Revision, 1948.
†N.S. = Not significant.

Only in the decade 55 to 64 did a possibly significant negative correlation ($p < 0.05$) appear for cirrhosis of the liver. In the case of motor vehicle accident deaths a high *positive* correlation was found; for other accidental deaths it was also positive but not as significant (Table I). The correlation for congenital malformations, at all ages, was also positive ($p < 0.01$). Correlation coefficients were small in the cases of other major causes of death, e.g., "certain diseases of early infancy," influenza and pneumonia and total infant deaths per 1,000 live births (Tables I and II).

DISCUSSION

There seems to be clearly established a relationship between weighted average hardness of "finished" water and average annual death rates by states in 1949 to 1951 for diseases associated with arterial hypertension and disorders secondary to atherosclerosis. The high correlation coefficients, -0.5 or more, for two of these disorders are significant at probability levels of about one in one thousand. Therefore, if one grants the validity of the data, this relationship can be accepted. Lack of significant correlations between water hardness and three major categories of heart diseases—chronic rheumatic heart disease, "nonspecific chronic endocarditis and other myocardial degeneration" (which undoubtedly includes an unknown number of cases of arteriosclerotic heart disease), and "other diseases of heart"—points further to a true relation between water hardness and atherosclerotic and/or hypertensive disorders.

Sauer and Enterline[6] recently examined a number of factors which might affect data on death rates and concluded that variations from state to state were real and were unaccountable by the quality or quantity of physicians. They also believe that population and total death rates are fairly accurate. The present study is based upon their conclusions and upon chemical and analytical data on specimens of municipal water collected during the period when the death rates were calculated (1949-1951). Errors in annual variations were thus largely avoided.

Among the noncardiovascular diseases, a possibly significant correlation with malignant neoplasms appeared only in respect to male respiratory tract neoplasia. Analyses of less common neoplasms included in the total were not made, for the death rates were based on too few cases for valid analyses. A suggestive relationship appeared for cirrhosis of the liver but for no other common diseases.

Of the ten leading causes of death, correlations were not statistically significant for diabetes mellitus, infant deaths, diseases of early infancy, or pneumonia and influenza, either for total population or older white persons, with the use of the rates available for 1949 to 1951 or for 1957. Curiously enough, a significant positive correlation did appear for congenital malformations in the total population and in white persons of all ages, which could not be explored further because of the low rates in adults. Rates for other diseases were too low in many states for accurate computation.

The curious positive correlation between water hardness and motor vehicle accident deaths may be a geographical accident. The hard water areas are in general those midwestern and mountain states with large areas and low densities

of population, where long-distance driving is usual. In many, motor vehicle laws are less strict than in the more crowded seaboard states; perhaps this factor accounts for the correlation. In order to explore this factor, a correlation coefficient was determined on total cardiovascular deaths in older white males by state and density of population per square mile: $r = +0.279$ ($p = 0.05$). The coefficient for hardness of water and density of population was -0.292 (< 0.05). These coefficients are much lower than that for water and cardiovascular death rates (-0.56).

It was impossible to compare death rates of white and nonwhite persons, since the cases were few in many states and in 17 there were few nonwhite persons, making computations invalid.[2] In order to include as many disorders as possible, death rates for persons in the decade below mandatory retirement age were chosen when the values were considered sufficiently accurate from a statistical standpoint.[2]

To ascertain the effects of the inclusion of deaths in nonwhite persons and errors therefrom on these correlation coefficients, calculations were made for all categories of diseases listed. Comparisons of white and total populations showed relatively minor changes in r values for most of the cardiovascular diseases; values became statistically significant only in the case of chronic nephritis when nonwhites were included. Those for several but not all malignant neoplasms became significant, as did those for diabetes mellitus, pneumonia in persons aged 55-64, and general arteriosclerosis, reflecting the higher incidences of these disorders in nonwhite persons from "soft-water" states. Therefore, unless both white as well as total populations are analyzed, errors due to this factor can appear if death rates differ in the two racial groups.

Yerushalmy and Palmer[7] proposed certain procedures for evaluating etiological and accessory factors in chronic diseases based on modifications of Koch's postulates. No inference as to etiological factors is intended in this report, and the influences associated with hardness of water are obviously accessory environmental factors at the most. However, some of the postulates of these authors are partly satisfied by this study, and some are not. In discussing this matter, it must be emphasized that correlation coefficients shown in the tables, while statistically significant, are usually small. In Tables I and II there are only four values each greater than 0.45. Yerushalmy and Palmer list the following points:

"1. The suspected characteristic must be found more frequently in persons with the disease in question than in persons without the disease, or

"2. Persons possessing the characteristic must develop the disease more frequently than do persons not possessing the characteristic."[7]

In so far as death rates in white persons are concerned, an *influence* associated with hardness of water is found at ages 55-64 in the cases of hypertensive heart disease and coronary heart disease, and at all ages in motor vehicle accidents and congenital malformations. This is only an influence, for there were no "persons without the disease" in any state or city, although there were many, perhaps, without "the characteristic."

The third point of these authors, concerned with testing the association for validity by investigating the relationship of the characteristic with other diseases,

has been investigated and little or no relationship can be found as far as this *influence* is concerned. Four causes of death stand out; smaller coefficients of correlation, while still considered statistically significant, may or may not be of actual significance. A coefficient of 0.45 is high in any study with such a large number of possible variables as this one has.

This report serves to emphasize a relationship of certain chronic disorders with a variable environmental factor to which all persons are exposed. There may be a cause-and-effect relation, i.e., some factor in, or not in, different waters influences these death rates, or each of the two parameters may be related to a third. In any event, experimental work along these lines may be rewarding.

SUMMARY

Further inquiry into the relationship between hardness of finished potable water by state and death rates from the ten leading causes of death and subdivisions thereof in white persons 55 to 64 years of age disclosed:

1. A. Negative correlations with hypertensive diseases and with common conditions secondary to atherosclerosis ($p < 0.01$).

B. No significant correlations with other diseases of the heart.

2. A. A possibly significant negative correlation ($p = 0.02$) with neoplasms of the male respiratory system.

B. No significant correlations with other common neoplasms.

3. A. A suggestive negative correlation ($p < 0.05$) with cirrhosis of the liver.

B. No significant correlations with diabetes mellitus, influenza and pneumonia, infantile disorders, ulcer of stomach or duodenum, "senility," or intestinal obstruction.

4. Positive correlations with motor vehicle accident deaths ($p < 0.01$) and congenital malformations ($p < 0.01$) in all age groups.

In hardness or softness of potable water may lie a clue to an influence affecting death rates from hypertension and atherosclerosis, and possibly a few other chronic disorders.

REFERENCES

1. Schroeder, H. A.: Relation Between Mortality From Cardiovascular Disease and Treated Water Supplies, J.A.M.A. **172**:1902, 1960.
2. National Office of Vital Statistics. Death Rates for Selected Causes by Age, Color, and Sex, United States and Each State, 1949 to 1951, Vital Statistics vol. 49, Special Rep. Nos. 16-27, 30-43, 48, 52, 58-60, 1959.
3. Herdan, G.: Statistics of Therapeutic Trials, Amsterdam, 1955, Elsevier Publishing Company.
4. Lohr, E. W., and Love, S. K.: Industrial Utility of Public Water Supplies in the United States, 1952; Parts 1 and 2, Geological Survey, Water-Supply Papers 1299 and 1300, Washington, D. C., 1954, U. S. Government Printing Office.
5. Statistical Abstract of the United States, ed. 80, U. S. Department of Commerce, Washington, 1959.
6. Sauer, H. I., and Enterline, P. E.: Are Geographic Variations in Death Rates for Cardiovascular Diseases Real? J. CHRON. DIS. **10**:513, 1959.
7. Yerushalmy, J., and Palmer, C. E.: Methodology of Investigations of Etiologic Factors in Chronic Diseases, J. CHRON. DIS. **10**:27, 1959.

SUICIDES IN MEDICAL AND SURGICAL WARDS OF GENERAL HOSPITALS

ROBERT J. STOLLER, M.D., AND FLOYD M. ESTESS, M.D.

LOS ANGELES, CALIF.

From the Department of Psychiatry, University of California School of Medicine, Los Angeles

(Received for publication April 28, 1960)

VOL 12 196

THE occurrence of suicides in persons admitted for treatment of somatic illnesses to medical and surgical wards of general hospitals constitutes an infrequent, though serious, problem. The medical literature has given little attention to this phenomenon. Pollack[1] has reviewed the charts of 11 such patients in one hospital. He noted that only one patient on admission was considered to suffer from a possible psychiatric disorder; all suffered from medical problems requiring evaluation and treatment in a general hospital, although 9 of the 11 were found by him to have had a "significant neuropsychiatric history." In only one history was there noted anything to suggest a threat of suicide. Seven of the patients showed signs of acute brain disorder; one showed signs of psychotic depression. In retrospect, evidence of severe emotional disorder appeared in 10 of the 11 patients at least 24 hours before suicide. Severe dyspnea was considered a significant precipitating event. Pollack concluded that: "(a) patients in a general hospital were not generally considered psychotic unless constantly, grossly, and visibly disturbed in behavior; (b) there appeared to be an unfamiliarity with the clinical picture of organic confusion states and the toxic psychotic reactions associated with alcoholism; (c) the suicide hazard had not been closely identified with organic psychosis by the nonpsychiatric physician, who showed a low index of suspicion of suicide in general hospital patients; and (d) psychiatric orientation among nonpsychiatric physicians generally appeared to be low."

Friedman and Cancellieri[2] reviewed 272 patients admitted to a general hospital after a suicide attempt (i.e., even more openly emotionally ill than Pollack's patients, though not suffering, as a group, from severe physical diseases). They noted that none made another attempt while in the general hospital. They disagreed with Pollack's conclusion that older patients with physical disease plus an organic psychotic reaction are suicidal risks; another review in the same paper of 606 hospitalized patients with chronic brain disease revealed no suicide attempts.

The present authors have collected the medical records of 33 patients (included are Pollack's patients) who committed suicide while hospitalized in medical

or surgical wards for nonpsychiatric conditions in 2 large public general hospitals located in a large metropolitan area from 1946 through 1955. Both of these institutions have extensive training programs, including available psychiatric facilities. Our material included the complete medical record, the autopsy findings, if an autopsy was performed, and the results of any official investigations. Although such records present many limitations in evaluating the nature of the phenomena under consideration, certain pertinent material was available in each of the charts.

DESCRIPTION OF SUBJECTS

The number of patients studied was 33, 18 from one hospital and 15 from the other.

At Hospital 1, the total admissions for the 10 year period were about 100,000. At Hospital 2, the total admissions for the 10 year period were about 700,000. Thus the incidence of successful suicide in Hospital 1 during this period was 15 per 100,000, while in Hospital 2 it was 2.5 per 100,000.

Thirty-two of the suicides were committed by men; only one by a woman. (Fifteen of the patients are from a hospital that allocates 2 to 3 per cent of its beds for women, the other hospital using 55 to 60 per cent of the beds for women.)

The oldest patient was 76, the youngest 28; the average age for the group was 58 years. This can be compared to the mean age of persons who committed suicide in the Los Angeles area, which is 48.9. Only 3 patients were under 40; over half were 60 or older.

Twenty-eight were white; 4 were Negro; 1 was Oriental.

Twenty were Protestant; 7 Catholic; 2 Jewish; 1 Buddhist; 3 had no religion stated.

Eleven patients were married; 11 single; 6 divorced; 5 widowed. It is to be noted that two-thirds of the patients were not married at the time of suicide.

Eight patients had wives living in the community; 9 had siblings; 3 had children; 2 had parents; 3 had other relatives. Eight, however, were without any relatives in the community.

Eleven of the patients were unskilled laborers; 13 semiskilled laborers; 4 were unemployed because they were institutionalized for nonpsychiatric chronic disease; 3 were retired or unemployed. One patient was a student; the employment of one other was unknown. It is of interest that no skilled or highly trained individual is represented in the group, and that in general these patients were clearly from a lower economic class. This reflects the public nature of the hospitals involved.

All of the patients had physical illnesses requiring hospitalization. Some had more than one major disease. We have recorded here only the more severe illnesses. Seven patients suffered from advanced states of cancer of the internal organs; 8 patients had far-advanced pulmonary tuberculosis. Four were in acute heart failure; 3 had severe hypertension; 4 had central nervous system syphilis; 2 had severe bronchial asthma (not related to other pulmonary disease); 2 had peptic ulcer; 8 had various other significant illnesses. Two-thirds of the patients were in considerable danger to their lives from an existing illness.

Two-thirds of them had been suffering from severe illness for more than 6 months; only 9 had been ill for less than the 6 month period.

Fourteen of the patients had been hospitalized for less than 10 days; 6 for more than 10 but less than 30 days; 13 for more than 30 days. Thus, two-thirds of this group had been hospitalized and observed for more than 10 days before their suicide.

Sixteen of the patients jumped to their deaths; 15 died of lacerations; 2 hung themselves. These figures take on special significance when broken down between the two hospitals involved. At Hospital 1, 14 patients jumped; while at Hospital 2, only 2 jumped. On the other hand, 14 patients at Hospital 2 died of lacerations, while only 1 did so at Hospital 1. Both hangings occurred at Hospital 2.

From 11 o'clock in the evening until 7 the next morning, 15 patients committed suicide; 7 did so from 7 P.M. to 11 P.M., 11 between the hours of 1 P.M. and 7 P.M.; no patient did so from 7 A.M. to 1 P.M. It is to be noted that two-thirds of the patients committed suicide during the night hours, and that the later mornings hours seemed to be a safe period.

Twelve of the records contained notations describing emotional disorders of apparently nonpsychotic proportions; 7 showed alcoholism, of whom 2 had alcoholic psychoses; 7 showed other organic psychoses; 2 charts contained descriptions of clear-cut paranoid psychoses; 5 charts contained no notations suggesting mental or emotional disorder. Thus, seven-eighths of these patients had overt mental or emotional problems recorded in their medical records prior to suicide.

For this entire group, 6 psychiatric consultations had been requested, of which 3 were performed. Of those consultations requested but not performed, in one case the patient committed sucide on the day of the request; in the other 2, several days had passed between the time of request and the patient's suicide. Of the 3 consultations that were performed, one case study was in progress, and in the other 2 the psychiatrists were not sufficiently impressed by the danger of suicide to comment on it.

Six of the patients made suicidal threats that were recorded by medical or nursing staff; of these 6, only 3 had psychiatric consultations requested after the threat.

Nine patients were noted by the staff to be depressed.

About one-half of the patients (7) at one hospital showed clinical signs suggestive of organic brain syndrome, while in about one-half (9) of the cases from the other hospital suggestive evidence was noted in the physicians' or nurses' records (e.g., "disoriented," "fell out of bed—senile," "stuporous"). At the one hospital where routine adequate postmortem examinations were made, it was found that one-third of the cases showed some kind of lesion of the central nervous system.

CASE REPORTS

Case 1.—A 63-year-old unemployed man entered the hospital after having been found unconscious 3½ hours previously by his wife. His history revealed that he was unemployed since

1946 because of "nervousness" and mucous colitis. In 1949 he first experienced anginal pain. In the month before entry his symptoms had become increasingly severe. For several weeks he had been very depressed. He was thought to have taken large amounts of barbiturates. The patient was admitted on an emergency basis after he had apparently died in the reception room. There, artificial respiration, intracardiac Adrenalin, and intravenous Coramine with oxygen seemed partly to revive the patient; intravenous fluids, and intra-arterial plasma were given. Endotracheal intubation and suction, oxygen per catheter, intravenous Coramine and Neosynephrine, as well as other medication, additional plasma, and whole blood, were administered. Within 48 hours the patient had been restored to physiologic homeostasis. On the sixth hospital day he was noted, for the first time, to express ideas of persecution and other delusions. At 1:30 A.M. on the seventh hospital day the patient opened the window near his bed and jumped out. He was found dead on the ground below shortly thereafter.

Case 2.—This 50-year-old white, divorced man, a laborer, had suffered from pulmonary silicosis in the past and was hospitalized at this time for far-advanced tuberculosis, with pulmonary hemorrages and dyspnea observed during the hospitalization. He was not noted at any time during his hospitalization to be depressed, to have made suicidal threats, or to have shown any other evidence of emotional distress. Just before committing suicide, the patient was observed to be suffering from "extreme apprehension," immediately after which he cut his throat with a razor and died.

Case 3.—This 70-year-old white, widowed man, a stagehand, had complained of dyspnea for 20 years, with 8 weeks of edema of the legs and feet, plus increase in dyspnea during this time, as well as substernal pain for 12 years. He was "verbose" and "confused" at night during the 7 days of his hospitalization. Prior to his suicide attempt, nurses' notes state: "Seems to be confused. Talks out loud disturbing other patients. Side rails on bed." He fell out of bed over these rails one night just after having been noted by the nurse to be "confused."

The doctor's notes on the day of the suicide attempt say: "Since patient's entry on ward he has repeatedly disregarded advice of staff, even when told what his actions would be upon his well being [sic]. During his stay to date on this ward, this patient has given no indication that he contemplated such an action" (suicide).

The patient during that evening stabbed himself in the left upper quadrant, followed by sudden marked heart failure. He died 2 days later in heart failure, being considered to present too great a risk for exploratory operation. Postmortem revealed no perforation of the intestine. Gross or microscopic examination of the brain is not reported.

DISCUSSION

These patients were mostly older, white Protestant men of lower income, suffering from severe, chronic, distressing physical illnesses. Almost all were noted to have suffered from overt psychiatric problems prior to suicide, though in only a few were these problems judged severe enough to warrant psychiatric consultation. While less than one-third were noted to be depressed before suicide, almost one-half had signs suggestive of organic brain disease. In this group of 33 patients, all of whom suffered from physical illness requiring hospitalization, 28 were noted to have overt mental or emotional problems mentioned in their medical records prior to suicide.

We do not know why almost all these patients were men (Table I). One might speculate that it is of crushing significance for a man to find himself no longer manly, unimportant in work, unwanted by family, unknown and unseen by others, lost in the mechanics of the ward. It is worth remarking that these are not the same kinds of people who commit suicide in psychiatric hospitals. The latter are driven primarily by inner fantasies that do not derive from present realities. Not so with most of the patients in this series, whose loss of hope is too

TABLE I. SUICIDE RATES PER 100,000 FOR THE UNITED STATES*

AGE	TOTAL MALE	TOTAL FEMALE
20–24	9.1	2.6
30–34	14.3	5.2
40–44	22.4	7.0
50–54	32.0	8.5
60–64	37.5	9.4
70–74	47.6	6.6
80–84	60.7	5.0
Over 85	56.9	4.3

Note: Men predominate in all age groups and the rate for women decreases in the older age groups.
*The metropolitan Los Angeles rate is similar.

VOL. 12 1960

close to a correct evaluation of their situation. It is, in fact, true that no one wants them, and not even organic brain disease dulls that awareness.

That neither physicians nor nurses were oriented to observe and write down significant psychiatric signs and symptoms is clear from the charts. The hospital chart, which should be an important means of insuring communication of informations regarding patients' treatment, did not always function this way in the charts we reviewed. When one considers that the personnel on these wards were in general not trained in handling psychiatric patients, the significance of their being aware of psychiatric disturbance sufficiently severe to warrant notation is even more striking than had such awareness occurred in psychiatrically trained personnel. In only 5 charts were there no notations suggestive of mental or emotional disorder, and one could suspect that personnel more experienced in emotional disorders might have noted what is not present in these charts. On a number of occasions, nurses' notes giving clear-cut evidence of depression or even of suicidal thoughts appear not to have influenced the physician, possibly because the nurses' notes were not read.

We are making two assumptions based on our experience which we think are demonstrated by the charts but which we cannot prove: (1) that skimpy, inadequate workups and progress notes reflect, among other things, a relative lack of interest or ability to observe psychopathology, and (2) that when there was no progress note by a physician referring to a remark in the nurses' notes regarding psychopathologic manifestations, this was the result of either not reading the nurses' notes or not caring what was noted. Such a situation might be compared to the physician on a psychiatric ward failing to respond to a nurse's note that the patient had developed a fever, or, on discovering it himself, not to consider it important enough to investigate further or to comment on it in the chart.

Motto and Greene[3] have discussed the physician's role in suicide prevention. They state, "At least 40 per cent (74) of the suicides and 60 per cent (116) of the attempted suicides [these were not inpatients] were under medical care or had been so within the prior six months. . . ." "Of the entire group, 84 (23 per cent) had been in touch with a physician for at least a month before the act. In 59 (16 per cent) instances the suicidal act occurred within a week of the last medical

contact, and in 14 (40 per cent) on the same day the doctor was seen." In our series, all patients were seen by one or more physicians on the day of suicide.

A history of previous attempts is to be taken seriously.[3,4] That none of the patients in our series gave a history of previous suicide attempt is perhaps due to the physicians' not asking. We cannot guess whether any of the patients had ever thought seriously of or attempted suicide before.

An important area in the prevention of suicide (as well as in the whole of the practice of medicine) is the problem of adequate communication among personnel. Since the aides and nurses are observing the patient more continuously than the doctors, their observation of the patient's behavior throughout the day and night should be of vital concern to the physician, though unfortunately this is frequently not so. When the doctor shows little interest in the nurse's observations, the nurse is not likely to go out of her way to present them to the physician, either in writing or by talking with him. Likewise, if the aide is not felt to be part of the treatment team, he is very unlikely to communicate to the nurse information which may be essential to the patient's life.

L.
2
60

In a few of these cases there seems to have been no indication of a potential for suicide; these cases must, if one has only the charts to go by, be considered as unpreventable. Of these 33 patients, some were in terminal condition and would inevitably have died not long after, had they not committed suicide. Nonetheless in our series the lives of a considerable number of patients would have been prolonged greatly had signs and symptoms of depression and suicidal intent been recognizable to the personnel and had personnel who were capable of recognizing these been able to communicate them adequately to the physician. Since many patients who seriously attempt suicide would be successfully treated by psychiatric methods if the suicide could be prevented, one cannot fail to be struck by the fact that in these hospitals, one of whose primary functions is the preservation of life, preventable death due to suicide on some occasions has not been prevented.

In view of the large number of our patients suffering from serious illness, and the fact that certain of them suffered from advanced stages of these illnesses, it would seem indicated that the training of aides, nurses, and physicians include some introduction to the problems of depression and suicide. It is apparent that the first line of defense against the serious suicide attempt in the general hospital is the sensitivity of the personnel to the possibility of its occurrence.

Worthy of note in this regard is the fact that the patient who combines signs of confusion due to organic brain disease with depression should provoke special caution. The patient confused by organic brain disease has even less judgment than those suffering from many other emotional illnesses, and sudden suicide, without warning, is frequent enough that the possibility should be kept in mind by those treating these patients.

In addition to the vital role played by the personnel in preventing suicides, the physical structure of the hospital itself is also significant. In our series, it is interesting to note that in the hospital where there was an obvious vantage point for jumping, 14 patients jumped, while at the other hospital where no such opportunity was easily available, only 2 jumped. At the latter hospital, 14 of the patients died of lacerations, while only one did so at the former hospital. It is

not necessarily true for certain types of individuals that when one form of suicide is prevented, another will inevitably take its place. Every effort at preventing suicide will cut its rate or delay the attempt, and the fewer opportunities there are available, the more delay is introduced into the problem. It is just this delay which is so vital, for its permits personnel to become aware of the depression and agitation and to seek appropriate consultation and management of the patient. With adequate psychiatric treatment, a large number of patients who were contemplating suicide will no longer feel so inclined. In view of the large number of patients in our series who died by jumping, it is recommended that architects keep this possibility in mind when constructing the upper floors of hospitals, not allowing some area of the hospital to become a focal point for suicides. On the other hand, personnel should be alert on medical and surgical wards to the care of sharp objects in order to minimize the use of these by patients.

VOL. 12 1960

We have no information that would adequately explain the difference in suicide rates in these 2 hospitals. We can speculate that because the hospital with the higher rate has a clear point of vantage for jumping, a method quick and easy to find is available to the patient planning suicide. Not having this simple technique available therefore forces the patients in the other hospital to a more devious and more cautiously planned technique. Delay is thus introduced, and may create the difference in rates. There is no evidence that the personnel in one hospital are more skillful in preventing suicide than in the other (e.g., that they are more carefully chosen, more highly motivated, better trained to detect depression, watch patients more closely, etc.).

It is also suggested that when a suicide attempt has been made, whether it is successful or not, there be conducted what Shneidman and Farberow[5] have described as "psychological autopsy" involving all personnel connected with the patient to determine what might have been done to prevent the attempt, what factors were involved in motivating the patient to suicide, what information some of the personnel may have had which they did not pass on to others, etc. If such inquiries can be held without taking on an accusatory aspect, the educational value to the personnel will be great and the preventive value for future patients will also be great. There would seem to be great benefit in making a complete analysis of the medical and psychiatric characteristics of the patients who commit suicide in any hospital, including the methods used, the time, place, the atmosphere of the ward the patient is in, etc. This might be done by a Suicide Prevention Committee functioning in all hospitals as a research team, the effect of which in the immediate handling of patients will also be apparent.

The statistics mentioned earlier call to mind a lonely old man, sick and in great distress, fearful that his life may be ending with nothing to show for it or to leave behind, with the expectation (rightly or wrongly) of a lingering death in the ward's anonymous blankness. Above all, his suicide is the desperate cure for the terrifying emptiness of his life. For this patient, whose death is so much less the result of inner disturbance than, for example, in the typical psychotic depression of the manic-depressive, the lifting of the sense of hopelessness should be less difficult. An awareness that the patient is in despair must come first;

then sympathy, interest, and softening of the deadly routine of many busy wards will relieve this kind of hopelessness.

The program described earlier has its value not only in the prevention of this person's suicide, but also in regard to a problem we have not discussed, that of the patient who is discharged from the hospital and who commits suicide shortly after. If this patient showed manifestations of possible suicide intent while he was still in the hospital, it is the responsibility of the hospital to recognize such intent and take necessary preventive steps.

SUMMARY AND CONCLUSIONS

Thirty-three patients over a 10 year period in the medical and surgical wards of 2 general hospitals committed suicide. All but one were men; they were older than the mean age of those who commit suicide, and they had fewer relatives in the community than average. All had severe physical illnesses. About one-half killed themselves by jumping; about one-half died following lacerations. Most of the patients had overt mental or emotional problems recorded in their medical records prior to suicide.

It was noted that a program for the reduction of suicide rates in a general hospital should include: (1) training of personnel in recognition of depression and suicidal thoughts; (2) adequate reporting, both in the hospital chart and in discussions with the physicians in charge of the case, of the suspicion of such problems; (3) removal of opportunities for suicide in the construction of hospitals, in the handling of potentially dangerous articles on the ward, etc.; (4) an attempt to provide maximum relief from pain and anxiety to the severely ill and suffering patient who is depressed or hopeless about his physical state; (5) early and adequate psychiatric consultation; (6) adequate treatment for the depression and suicidal thoughts; (7) a Suicide Prevention Committee.

Many of these suicides are preventable if one is alert to the possibility of suicide and then eases the loneliness of these suffering people.

The authors are deeply indebted to Edwin S. Shneidman, Ph.D., and Norman L. Farberow, Ph.D., who have been of invaluable assistance in the preparation of this paper.

REFERENCES

1. Pollack, S.: *in* Shneidman and Farberow,[5] pp. 152-163.
2. Friedman, J. H., and Cancellieri, R.: Suicidal Risk in a Municipal General Hospital, Dis. Nerv. System **19**:556, 1958.
3. Motto, J. A., and Greene, C.: Suicide and the Medical Community, A.M.A. Arch. Neurol. & Psychiat. **80**:776, 1958.
4. Elias, R. B.: Is Suicide Preventable? California Med. **90**:128, 1959.
5. Shneidman, E. S., and Farberow, N. L.: Clues to Suicide, New York, 1957, McGraw-Hill Book Company, Inc.

THE OCCUPATIONAL-SOCIAL CLASS RISKS OF CANCER MORTALITY IN MEN

PHILIP BUELL,*
JOHN E. DUNN, JR., M.D.,**
AND LESTER BRESLOW, M.D.***
BERKELEY, CALIF.

From the California State Department of Public Health
(Received for publication March 20, 1960)

VOL
12
196

THE interpretation of the occupational mortality in England and Wales during 1910 to 1912 was that many causes of death among men were probably influenced more by the general conditions of life implied by an occupation rather than directly by the risks in the work itself. Before 1910 the data had been analyzed only for direct occupational risks. After that time, the deaths among married women, analyzed according to occupation of husband, showed similar differences by social class. Social class gradients were found for a number of diseases including cancer of several sites.

The cancer death records of California men for the years 1949 to 1951 can be analyzed in much the same way by assigning the death certificates to one of five social class levels according to the reported occupation; for comparison, the most recent British report is for approximately the same period, 1949 to 1953.[1] Another useful comparison is with the cancer incidence variations by income level of Census tracts in ten United States cities (the Ten City Survey),[2] which offers a check on consistency between social class mortality and morbidity from cancer. Unfortunately, the death certificates of California women did not carry the occupational information on husbands until 1958.

METHODS AND MATERIALS

An earlier report on coronary heart disease[3] shows in detail the source, and some limitations, of the occupational mortality data for 1949 to 1951. The present paper deals with 10,401 cancer deaths occurring in that period among men aged 20 to 64. Each male decedent was assigned to one of five social class levels according to the occupation reported on the death certificate. The Census enumerations of occupations in 1950, plus some unpublished tabulations, provided the

*Associate Public Health Analyst, Bureau of Chronic Diseases.
**Field Investigations and Demonstrations Branch, National Cancer Institute, detailed to California Department of Public Health, Bureau of Chronic Diseases.
***Chief, Bureau of Chronic Diseases.

populations at risk by age and social class. The numbers of deaths to expect in each social class, if they do not differ from one another in cancer mortality, were then computed according to the age and cause specific rates of mortality for all classes. The ratio of the observed to the expected deaths, with the latter adjusted for age, shows the variations in risks. Expressed as a percentage, this ratio becomes the Standardized Mortality Ratio (S.M.R.), 100 for all men with civilian work experience and slightly less for all men with reported occupations.

The social class levels consist of five occupational levels developed by the National Office of Vital Statistics for grouping Census occupations, as discussed earlier.[3] The five levels represent only intuitive judgments about common conditions of life but may be adequate for detecting gross differences in social class mortality.

The numbers of men in each social class are shown in Table I. The proportions by social class differ slightly between California and England and Wales, but in both areas the classes at each extreme (professionals and unskilled workers) are small in proportion to the others; this will tend to maximize mortality differences that might otherwise be obscured with a crude measurement of social-environmental level. A major difference should be noted: in California, farmers and farm laborers are excluded from the classification for reasons noted later; in England and Wales, farmers are in Social Class II and most farm laborers in Social Class IV. Another difference is the composition of Social Class I: in England and Wales this class consists of both the professions and the higher officials and higher business groups; in California only the professions are included while all officials and business groups are in Social Class II because of inability to make fine distinctions in the data.

In the British report[1] the social-environmental circumstances of an occupation are considered to be the locality, housing, recreation, dietary habits, attitudes

TABLE I. DISTRIBUTION OF THE MALE POPULATION, AGED 20 TO 64, BY SOCIAL CLASS CALIFORNIA AND ENGLAND AND WALES, 1950

SOCIAL CLASS		CALIFORNIA		ENGLAND AND WALES
		NO.	%	%
I	Professionals, etc.	151,802	5.1	3.4
II	Intermediate	523,021	17.5	15.2
III	Sales, clerical, skilled	1,058,540	35.5	51.9
IV	Semiskilled	657,552	22.0	15.7
V	Unskilled	295,961	9.9	12.3
	Farmers	100,572	3.4	*
	Farm laborers	156,751	5.3	†
	Occupations not reported	40,668	1.4	
	Total	2,984,867	100.1	98.5

*Farmers included in Social Class II.
†Most farm laborers included in Social Class IV.

toward health, etc., of those in the occupation. Income, used for the Census tract data in the Ten City Cancer Morbidity Survey, is not used in assigning the occupations, either in Britain or California, but certainly something of this nature is also implied. One could add that innate or early acquired health characteristics may also be correlated with social class or income. Obviously, if one finds an association with a disease, it serves only to raise multiple questions about the factors that may be responsible for it.

The individual occupations and occupational groups we were able to examine consisted of 106 titles, exclusive of farmers and farm laborers. These are not in sufficiently fine detail for a thorough analysis of occupational and industrial risks of cancer, though specific occupations will be mentioned where it seems warranted.

OCCUPATION REPORTING

Death and census records are not completely in agreement in listing occupations, and, in fact, there is an apparent inconsistency between them. The death certificates in 1949 to 1951 requested *usual* occupation while the Census enumerated *last* occupation in 1950. This problem has already been touched on in a study[4] which compared death certificates with detailed occupational histories taken before death; despite the request for *usual* occupation, defined as longest held, last occupation was found to be reported more often on the death certificate. We have used the data from that study to estimate, roughly, the reporting bias (between last occupation and that entered on the death certificate) for each social class. This is shown in Table II. The data are not representative of all state death

TABLE II. DEATH CERTIFICATE OCCUPATION REPORTING BIAS AS IT AFFECTS SOCIAL CLASS GROUPINGS

SOCIAL CLASS	NUMBER FROM OCCUPATIONAL HISTORY (1)	NET EFFECT OF BIAS		ENTRY ON DEATH CERTIFICATE SAME AS FROM OCCUPATIONAL HISTORY†	
		NUMBER ON DEATH CERTIFICATE (2)	PER CENT* (3)	NUMBER (4)	PER CENT (5)
I	11	14	127	11	100
II	26	26	100	21	81
III	113	122	108	99	88
IV	91	72	79	59	65
V	47	48	102	35	74
Farmers	6	11	183	5	83
Farm laborers	10	4	40	3	30
Unknown	0	7	—	—	—
Total	304	304		233	

*Ratio of number reported on death certificate to number ascertained by history (columns 2/1 = 3). Column 3 suggests the variation of an S.M.R. from a value of 100 due to occupation reporting bias.

†The ratios shown in column 5 (4/1 = 5), will suggest whether an actual S.M.R. reflects all that it should; for example, if the S.M.R. for a disease in Social Class V is greater than 102 (column 3), only 74 per cent (column 5) of the true excess is reflected in the S.M.R.[4]

certificates and we use them only to judge the direction of possible reporting bias. Column 3 suggests a slight, but not uniform, "upgrading" of occupations on the death certificates, with overreporting in Social Class I, and underreporting in Social Class IV, but not, it should be noted, in Social Class V.

Generally, the estimates of misreporting suggest the following effects on mortality gradients: inverse associations, i.e., gradients increasing from Social Class I to V may be minimized or obscured; direct associations, i.e., gradients decreasing from Social Class I to IV may be due, in whole or in part, to the influence of misreporting. There is no evidence of occupation reporting error in Social Classes II and V.

Farmers and farm laborers were considerably underrepresented in the occupation accuracy study so that estimates of misreporting are based on quite small numbers, as shown in Table II. The evidence, such as it is, suggests that bias greatly increases the S.M.R.s for farmers, and considerably diminishes the S.M.R.s for farm laborers. Because of this possibility of serious reporting error, the discussion of these two occupations is separate from the main report. It should be added that the Buechley study[4] mentioned earlier did not find that the excess of reported farmers came from the unreported farm laborers. The misreporting was more complex.

Somewhat the same occupation reporting disagreements are shown for England and Wales,[1] although they are based on a comparison of death records and Census records rather than death records and interview histories. The reporting discrepancies overstate the mortality in Social Classes I and II and diminish slightly the mortality in Social Classes IV and V.

RESULTS

Gradients Increasing From Social Class I to Social Class V.—Deaths from cancers of all sites, shown in Table III, increase slightly, and somewhat irregularly, with lower social class. Misreporting of occupations, it was pointed out, tends to obscure gradients in this direction and may be the cause of the irregularity in the gradient.

Sites of cancer which reveal a definite inverse association with social class in California are found in the upper alimentary canal: buccal cavity and pharynx, esophagus, and stomach (Table III). The latter two show strong and quite regular gradients. The association with buccal cavity and pharynx combined is not as strong, showing no unusually high social class risks from these cancers, but considerable protection for Social Classes I and II, especially for the former with an S.M.R. of 67.

Lung cancer mortality also increases roughly from Social Class I to Social Class V though the association is not as strong as that for cancers of the upper alimentary tract. The lung cancer gradient is irregular, but this too may be caused by occupation misreporting.

Gradients Decreasing From Social Class I to Social Class V.—Several sites of cancer show an apparent direct association with social class but, in all except one, the trends are slight and the possibility is strong that "upgrading" or other discrepancies in reporting occupations on death certificates have caused the

TABLE III. STANDARDIZED MORTALITY RATIOS, CANCERS OF SELECTED PRIMARY SITES, BY SOCIAL CLASS
CALIFORNIA MEN, AGES 20 TO 64, 1949 TO 1951

CANCER SITE (SIXTH REVISION INTERNATIONAL LIST NUMBER)	TOTAL NUMBER OF DEATHS	STANDARDIZED MORTALITY RATIOS							
		ALL REPORTED OCCUPATIONS*	SOCIAL CLASS†					FARMERS‡	FARM LABORERS‡
			I	II	III	IV	V		
All sites	10,401	98	97	90	103	97	112	104	62
Buccal cavity and pharynx (140-148)	440	95	67	80	102	112	100	104	56
Esophagus (150)	287	96	54	67	86	115	145	144	93
Stomach (151)	1,210	98	51	72	93	99	165	132	95
Intestine and rectum (152-154)	1,306	98	111	112	107	86	92	93	29
Pancreas (157)	568	98	93	88	100	113	97	123	60
Trachea, bronchus, and lung (162-163)	2,146	99	85	77	112	108	112	94	54
Prostate (177)	384	97	135	112	96	88	107	79	52
Kidney (180)	262	100	92	89	116	98	107	86	57
Bladder (181)	351	97	106	106	99	98	82	110	63
Brain (193)	454	98	130	127	108	77	58	100	52
Leukemia (204)	491	99	104	116	101	86	104	127	46
Lymphoma, etc. (202-203, 205)	728	98	164	100	101	88	107	94	42
All other sites	1,774	97	101	83	100	96	119	101	89

*S.M.R. for all men including unreported occupations is 100.
†Social Class I, professionals; Class II, intermediate; Class III, sales, clerical, and skilled; Class IV, semiskilled; Class V, unskilled.
‡S.M.R.s for farmers and farm laborers are subject to more serious reporting error (see text).

VOL. 12 1960

gradients. Gradients for cancer of the intestine and rectum (analyzed together) and bladder, showing trends downward from Social Class I to Social Class V, and for cancer of the prostate with a trend downward from I to IV, are suspect because the variations are similar to those which might be produced by occupation reporting bias (column 3, Table II). The Ten City Survey of incidence by income level, which is free from this bias, does not reveal clear evidence of associations for these sites (Table IV). On the other hand, the Registrar-General's data for England and Wales, also influenced by this reporting bias,[1] shows a moderate association for large intestine but not for rectum (Table IV). There is also, in England and Wales, a relatively greater mortality from cancer of the prostate in Social Class I, but no progressive gradient for this site, or for the bladder.

The brain, including other parts of the central nervous system, is the only site studied for which cancer mortality in California men shows a direct social class association that cannot be explained by occupation reporting bias. Whether some other source of bias may be present is taken up in the detailed discussion of separate sites of cancer.

Buccal cavity and pharynx: Several sites of cancer are included under the term "buccal cavity and pharynx" (lip, tongue, mouth, and pharynx), and the advantage gained in grouping these sites, that of getting sizable numbers for analysis, would be lost if the social class variations behaved differently for the separate sites. The Ten City Survey data show that only the salivary glands fail to follow the same general variations by income level for these sites. Less than 10 per cent of the incidence of oral and pharyngeal cancers were attributed to cancer of the salivary glands. In England and Wales the social class variation of persons suffering from cancer of the lip differed from social class variations for other oral sites at ages 20 to 64; lip cancer accounted for less than 5 per cent of this group between 1949 and 1953. These differences would not obscure trends for the major site group as a whole.

The Ten City Survey incidence data for cancer of the lip, tongue, mouth, and pharynx (Table IV) show gradients by income class generally consistent with the social class results for California, 1949 to 1951. The risk of cancer of these sites rises with decreasing income level. The comparison with mortality data for England and Wales, however, shows an unusual contrast. Table V depicts the results for oral and pharyngeal cancer as a group. While the inverse association with social class in California appears at ages 20 to 64 in 1950, it is present in England and Wales only in 1930 to 1932—and even then appears to be changing slightly at the younger ages (45 to 54). By about 1951 there is no longer the same relationship with social class, a change especially evident at ages 45 to 54 and 55 to 64. The younger cohorts in England and Wales fail to show the differential social class risk found earlier in that country and found in California in 1950. The exception may be cancer of the lip, which continues to show an inverse association, based on quite small numbers, in England and Wales.

Transfers of occupations between the social classes have occurred since 1931[1] but are quite unlikely to be responsible for the disappearance of the gradients as shown in Table V. Occupational errors, on the other hand, may have

TABLE IV. STANDARDIZED INCIDENCE RATIOS IN UNITED STATES CITIES AND STANDARDIZED MORTALITY RATIOS IN ENGLAND AND WALES FOR CANCERS OF SPECIFIC SITES AMONG MEN

PRIMARY SITE	TEN UNITED STATES CITIES, 1947 STANDARDIZED INCIDENCE RATIOS BY INCOME CLASS (ALL AGES)					ENGLAND AND WALES, 1949-1953 STANDARDIZED MORTALITY RATIOS BY SOCIAL CLASS (AGES 20-64)				
	I*	II	III	IV	V†	I‡	II	III	IV	V§
Lip	86	84	93	106	129	*33*	*60*	103	136	107
Tongue	61	65	123	114	118	106	104	101	89	102
Salivary glands	113	119	65	114	112	*150*	89	101	133	58
Mouth	69	93	78	134	130	127	93	94	113	106
Pharynx	74	95	94	91	144	122	100	98	101	100
Esophagus	55	88	105	102	137	132	92	91	98	130
Stomach	71	90	102	105	124	57	70	101	112	130
Large intestine	101	102	102	94	100	121	101	102	92	99
Rectum	92	108	99	97	104	86	86	108	94	104
Pancreas	102	84	96	102	119	120	101	101	93	103
Lung, bronchus	67	78	99	118	134	81	82	107	91	118
Prostate	88	106	96	98	112	128	99	102	93	102
Kidney	93	103	102	102	98	134	100	106	96	82
Bladder	113	103	97	93	100	106	77	109	96	107
Brain	114	92	91	105	108	133	96	104	88	99
Leukemia						123	98	104	93	89
Acute	105	112	92	92	105					
Chronic	119	79	91	110	107					
Other	104	89	119	103	76					
Lymphoma										
Lymphosarcoma	91	89	119	96	95	138	104	103	86	91
Hodgkin's	92	81	114	97	107	142	110	100	93	87
Other lymphoma	87	127	94	100	93	*113*	134	102	70	91
Multiple myeloma						188	101	104	92	76

*High income.
†Low income.
‡Professional workers.
§Unskilled workers.
Note: Standardized Mortality Ratios in italics are based on fewer than 10 deaths observed.
Source: U.S. data from Dorn and Cutler,[2] England and Wales data from Registrar-General's report.[1]

some effect; Logan[6] reported on a serious error introduced into Social Class I death rates because of lack of comparability between records of "company director" on death certificates and at Census. Deaths, from all causes, in this occupation totaled 1,414 (about 10 per cent of the total for Social Class I) against 98 expected. The majority of the excess should probably be assigned to Social Class II, but the effect on Class II would be small. Logan[7] points out that this error for company directors may be more serious for 1951 than for 1931 because of the increase in the number of small companies. If 10 per cent or more of the oral and pharyngeal cancers in Social Class I in 1949 to 1953 are due to inaccurately assigned deaths among company directors, then a high S.M.R. for Social Class I is questionable. But the masking of a true low S.M.R., as low as in 1931, is doubtful, and the true S.M.R. is likely to be about 100. In any case, the evidence

VOL. 12 1960

is strong from the remaining classes that the S.M.R.s are by 1951 more or less uniform where 20 years earlier they assumed a definite social class gradient.

From Case's cohort analysis[5] we know there has been a considerable decrease in the death rate due to oral and pharyngeal cancers in the four English male cohorts born around 1871, 1881, 1891, and 1901. This decrease must have affected the social classes differently in order to wipe out the gradient that existed in 1930 to 1932. Table VI shows that the percentage decreases are greatest where the rates were initially higher, i.e., Social Class V shows the greatest, and Social Class I the least, amount of decrease. There was, however, a substantial decrease in rate in each class.

There has been some decrease in the death rates for persons with cancers of the buccal cavity and pharynx among men in this country, but not to the extent found in England. The appropriate time-trend data for men in California are not published by age groups, and we use instead, in Table VII, some cohort data for white men in the United States to compare with similar data in England and Wales. (United States data are not available to show cohort death rates before 1930, nor are the age groupings as refined, but the trends are sufficient to indicate the main points.) The cohort born around 1871 in England and Wales shows much higher rates than the comparable cohort in the United States. Beginning with the 1890 cohorts, there is a complete shift and the rates for the United States exceed those for the comparable cohorts in England and Wales, not because of an increase in the United States but due to a considerably greater decrease in Britain. For example, the 1890 cohort in the United States has a rate of 16.0 per 100,000 at ages 55 to 64 while the same England and Wales cohort shows at ages 60 to 64 (a slightly higher average age) a rate of only 14.4. A similar difference is shown for the cohorts born around 1900. The differences would be greater if exact age group comparisons could be made.

While some decrease can be noted in the United States rates, they are stabilizing, temporarily at least, at a higher level than in England and Wales. The

TABLE V. MORTALITY FROM CANCER OF BUCCAL CAVITY AND PHARYNX. STANDARDIZED MORTALITY RATIOS FOR MEN BY SOCIAL CLASS IN CALIFORNIA, AND ENGLAND AND WALES

SOCIAL CLASS	CALIFORNIA	ENGLAND AND WALES*					
	1949 TO 1951	1930 TO 1932			1949 TO 1953		
	AGED 20 TO 64	AGED 45 TO 54	AGED 55 TO 64	AGED 65 TO 69	AGED 45 TO 54	AGED 55 TO 64	AGED 65 TO 69
I	67	104	63	65	135	113	100
II	80	79	62	65	95	96	83
III	102	91	93	107	100	99	103
IV	112	115	108	101	98	105	107
V	100	127	156	145	94	101	132

*For the source of data on England and Wales, see Registrar-General's Decennial Supplement, England and Wales (Occupational Mortality) 1931 and 1951, London, 1938 and 1958.

TABLE VI. DEATHS FROM CANCER OF BUCCAL CAVITY AND PHARYNX. RATES PER 100,000 MEN, BY SOCIAL CLASS IN ENGLAND AND WALES, 1930 TO 1932 AND 1949 TO 1953

	1930 TO 1932		1949 TO 1953		
	TOTAL DEATHS 3 YEARS	ANNUAL RATE	TOTAL DEATHS 5 YEARS	ANNUAL RATE	PERCENTAGE DECREASE IN RATES
45 to 54 Years					
Social Class I	26	12.3	21	4.3	65
II	116	9.3	76	3.1	67
III	333	10.7	224	3.2	70
IV	152	13.5	73	3.2	76
V	169	14.9	61	3.0	80
55 to 64 Years					
Social Class I	53	30.5	47	14.0	54
II	294	29.9	213	11.9	60
III	993	45.1	540	12.3	73
IV	471	52.3	218	13.0	75
V	719	75.6	216	12.4	84
65 to 69 Years					
Social Class I	37	59.0	39	30.1	49
II	193	59.8	159	25.0	58
III	678	97.6	496	31.2	68
IV	271	92.0	197	32.4	65
V	387	132.3	266	40.1	70

Source: See Table V.

stabilization is suggested by the similarity in the rates for the 1900 and 1890 United States cohorts at ages 45 to 54 and by the similarity of the 1890 and 1880 United States cohorts at ages 55 to 64. Given a stabilized rate and a social class gradient in oral and pharyngeal cancer mortality in 1950, one would predict a continued social class gradient at ages 20 to 64 when the 1960 social class mortality is analyzed in California. If this proves to be the case it will be especially noteworthy in view of the disappearance of the gradient in England and Wales by 1951. This is not to say that a social class gradient would necessarily disappear if the rate were reduced over time; but the British experience suggests that a reduction in cancers of these sites, which does not seem to be happening in the United States, would benefit mainly the poorer classes, which started with higher rates.

The 5 social classes in California consist of a grouping of 106 occupations. When these occupations were looked at in detail only 4 of 106 showed an excess of mortality from cancer of the buccal cavity and pharynx significant at the 0.05 probability level,* and none showed significantly fewer deaths than expected. A variation of 4 out of 106 can be expected on the basis of sampling variation alone. One of the 4, however, showed an unusual excess: musicians, with 8 deaths

*This significance level will be applied throughout. For an expected number less than 20 we used Table I by Cutler and associates[8] to determine significance. For larger numbers we took the square root of the expected number of deaths as an approximation of the standard error.

observed and 1.25 deaths expected. The other 3 occupations were: policemen, Social Class III; cooks, Class IV; and laborers, not on farms or in mines, Class V.

Esophagus: The risk of mortality from esophageal cancer increases sharply with lower social class in California, a result strongly supported by incidence rates from the Ten City Survey. On the other hand, as Table VIII reveals, the same association was found in England and Wales only in 1921 to 1923 and 1930 to 1932; by about 1951 the gradient was altered by a high risk, not only for Social Class V, but also for Social Class I which had earlier enjoyed considerable protection from the disease.

Just as with oral and pharyngeal cancer, there is the possibility that inaccurate occupation recording for company directors in the period around 1951, mentioned earlier, has pushed the S.M.R. for Social Class I out of line. Dr. Logan,[7] of the General Register Office, has kindly furnished some unpublished data on esophageal cancer in the separate occupations of Social Class I for the 4 years 1950 to 1953. If not only the company directors but also commissioned officers in the armed forces are removed, because their high S.M.R.s for all causes also raise doubts about occupation recording, the S.M.R. for Social Class

OL.
12
960

TABLE VII. DEATH RATES PER 100,000 FROM CANCER OF THE BUCCAL CAVITY AND PHARYNX IN COHORTS OF MEN IN THE UNITED STATES, AND IN ENGLAND AND WALES

AGE GROUPS		YEAR OF BIRTH							
UNITED STATES	ENGLAND AND WALES	UNITED STATES 1870	ENGLAND AND WALES 1871	UNITED STATES 1880	ENGLAND AND WALES 1881	UNITED STATES 1890	ENGLAND AND WALES 1891	UNITED STATES 1900	ENGLAND AND WALES 1901
25 to 34								0.3	
	25–								
	30–				0.5		0.4		0.4
35 to 44						1.6		1.1	
	35–				1.1		1.0		0.5
	40–		6.1		3.5		1.4		0.9
45 to 54				7.5		6.0		6.2	
	45–		14.3		8.5		3.5		1.9
	50–		26.4		13.4		5.8		4.3
55 to 64		19.0		16.5		16.0			
	55–		42.7		19.0		8.6		
	60–		54.9		27.5		14.4		
65 to 74		34.7		29.3					
	65–		65.0		32.8				
	70–		75.9		49.2				
75 to 84		52.5							
	75–		80.7						
	80–		101.5						

Source: Cancer Mortality for Selected Sites by Age, Sex, and Race: U.S. 1930 to 1945 (Vital Statistics—Special Report Vol. 32, No. 3); Vital Statistics of the United States, 1950, Vol. III; England and Wales data from Case.[5]

TABLE VIII. MORTALITY FROM CANCER OF THE ESOPHAGUS. STANDARDIZED MORTALITY RATIOS FOR MEN BY SOCIAL CLASS IN CALIFORNIA, ENGLAND AND WALES, AND COPENHAGEN

SOCIAL CLASS	CALIFORNIA	ENGLAND AND WALES					COPENHAGEN
	1949 TO 1951	1921 TO 1923	1930 TO 1932		1949 TO 1953		1943 TO 1947
	AGES 20–64	AGES 20–64	AGES 20–64	AGES 65–69	AGES 20–64	AGES 65–69	ALL AGES
I	54	76	75	46	132	100	104
II	67	91	87	86	92	90	103
III	86	104	103	106	91	99	67
IV	115	88	94	91	98	115	80
V	145	130	130	138	130	130	122

Source: England and Wales data, see Table V. Copenhagen data from Clemmesen and Sorensen.[9]

TABLE IX. DEATH RATES PER 100,000 FROM CANCER OF THE ESOPHAGUS IN COHORTS OF MEN IN THE UNITED STATES AND IN ENGLAND AND WALES

AGE GROUPS		YEAR OF BIRTH							
UNITED STATES	ENGLAND AND WALES	UNITED STATES 1870	ENGLAND AND WALES 1871	UNITED STATES 1880	ENGLAND AND WALES 1881	UNITED STATES 1890	ENGLAND AND WALES 1891	UNITED STATES 1900	ENGLAND AND WALES 1901
25 to 34								0.0	
	25–30				0.2		0.2		0.2
35 to 44						0.5		0.5	
	35–40		3.5		0.5 1.8		0.4 1.2		0.6 1.5
45 to 54				4.3		4.2		3.4	
	45–50		9.1 19.7		5.3 10.7		2.6 6.2		2.7 5.4
55 to 64		12.1		12.4		12.5			
	55–60		29.7 45.7		19.3 26.0		11.4 18.5		
65 to 74		22.6		25.5					
	65–70		56.0 62.1		37.0 47.5				
75 to 84		34.7							
	75–80		78.9 85.7						

Source: Cancer Mortality for Selected Sites by Age, Sex, and Race: U.S. 1930-1945 (Vital Statistics —Special Report Vol. 32, No. 3); Vital Statistics of the United States, 1950, Vol. III; England and Wales data from Case.[5]

I at ages 20 to 64 is reduced from 132 to 102, with the latter ratio based on 54 observed deaths for the 4 years 1950 to 1953. While the elevated risk disappears with the adjustment, the class does seem to have lost the relative protection enjoyed earlier as shown by the S.M.R. of 75 in 1930 to 1932. Also, the social class differences in England now tend to produce a "U" shaped distribution similar to that (Table VIII) reported for esophageal cancer in Copenhagen by Clemmesen and Sorensen,[9] who grouped subdistricts of the city by average rental values. This may not be unusual since the mortality from esophageal cancer has been decreasing both in Britain and Denmark, and Social Class I, having the lowest rates to start with, might be expected to experience the smallest percentage of decrease.

The decrease in mortality from cancer of the esophagus among all men in England and Wales, when social class is not considered, is shown in Table IX, taken from Case's data on cohorts.[5] In contrast, there have been no substantial changes in the rates for comparable United States cohorts, shown in the same table, and the British rates have come down to about the level of the United States cohorts of 1890 and 1900. The percentage decrease in the rate for each social class in England and Wales, from about 1931 to 1951, is shown in Table X. The age specific rates in the table cannot be adjusted for error in recording of company directors in Social Class I.

Because Social Class I in California does not include the higher business occupations, the relative protection from esophageal cancer shown by that class reflects low rates for the professions only. In England, the failure of Social Class I to show the same degree of relative protection is due largely to the inclusion of higher business and managerial occupations (excluding the forementioned company directors and commissioned officers), as shown in the unpublished data furnished by Dr. Logan.[7] While the differences are too small to give them significance in a statistical sense, these businessmen, plus engineers, authors, journalists, and publicists, had 35 observed and 25.7 expected deaths from esophageal cancer against 19 and 28.3 for the rest of Social Class I, largely professionals. This suggests a factor correlated with social class, but more closely associated with particular occupational groups. Clemmesen feels that this factor might be alcohol.[9] He notes that in Denmark the occupations "exposed" to alcohol (commercial travelers and persons in hotel and restaurant trades) have quite high mortality and incidence rates due to esophageal cancer. In England and Wales publicans and inn and hotel keepers had 38 observed against 16 expected deaths from cancer of the esophagus, and makers of alcoholic drinks had 6 and 2, respectively. Since the occupational detail was not fine enough, the only similar occupation we are able to observe in California is waiters and bartenders with 9 observed and 3 expected deaths. Four other occupations had statistically significant excesses for esophageal cancer, bakers and cooks in Social Class III, and janitors and longshoremen in Class V. Three occupations had significantly fewer deaths than expected: "other" clerical workers, machinists and job setters, "other" mechanics (not auto, airplane, or radio), all in Social Class III.

Stomach: The poorer classes have consistently shown a greater risk of death from stomach cancer in England and Wales in 1921 to 1923, 1930 to 1932, and

1949 to 1953. The gradient is generally quite regular and the ratio of Social Class V to I is twofold at ages 20 to 64. In California at these ages the same ratio in 1949 to 1951 is threefold. The ratio from the Ten City Survey morbidity data by income level is not as strong, only 1.7, but includes men of all ages. This social class differential seems to be maintained whether the stomach cancer mortality rates decrease or not. In 1930, as Haenszel[10] has shown, England and the United States had almost equal rates; by about 1950 the United States rate was reduced about one third and the rate in England and Wales remained almost stationary. When the social class mortality data are available for California in 1960 it will be possible to see whether the rate of decrease is constant for all social classes.

The analysis of specific occupations showed nine occupations with an excess number of deaths significant at the 0.05 probability level: bakers, masons, plumbers, and toolmakers, all in Social Class III; meat cutters, not in packing house, and cooks, in Social Class IV; lumbermen, laborers, and longshoremen in Social Class V. Significant deficits were found for accountants, Class I; teachers, Class II; managers, officials, and proprietors in trade, in manufacturing, and in "all other industries," Class II; "other" clerical workers, Class III. A significant variation of 15 out of 106 observations is a greater number than is likely to occur through sampling variation. While the variations for many of the 15 occupations fit a social class pattern, the high S.M.R.s for bakers, masons, plumbers,

TABLE X. DEATHS FROM CANCER OF THE ESOPHAGUS. RATES PER 100,000 MEN, BY SOCIAL CLASS IN ENGLAND AND WALES, 1930 TO 1932 AND 1949 TO 1953

	1930 TO 1932		1949 TO 1953		
	TOTAL DEATHS 3 YEARS	ANNUAL RATE	TOTAL DEATHS 5 YEARS	ANNUAL RATE	PERCENTAGE DECREASE IN RATES
45 to 54 Years					
Social Class I	24	11.3	26	5.4	52
II	84	6.7	82	3.3	51
III	228	7.3	247	3.6	51
IV	91	8.1	80	3.5	57
V	133	11.7	108	5.4	54
55 to 64 Years					
Social Class I	38	21.8	64	19.0	13
II	323	32.9	247	13.8	58
III	817	37.1	577	13.1	65
IV	316	35.1	240	14.3	59
V	441	46.4	309	17.8	62
65 to 69 Years					
Social Class I	18	28.8	41	31.6	(+10)
II	173	53.6	181	28.4	47
III	456	65.6	497	31.2	52
IV	167	56.7	221	36.3	36
V	250	85.5	272	41.0	52

Source: See Table V.

TABLE XI. MORTALITY FROM LUNG CANCER AND ALL CAUSES. STANDARDIZED MORTALITY RATIOS BY MAJOR OCCUPATION GROUPS IN CALIFORNIA MEN, AGED 20 TO 64, 1949 TO 1951

MAJOR OCCUPATION GROUP		LUNG CANCER			ALL CAUSES S.M.R.	OCCUPATION REPORTING BIAS†	
NUMBER	TITLE	DEATHS (1)	ACTUAL S.M.R. (2)	PREDICTED* (SMOKING) S.M.R. (3)	(4)	NUMBERS‡ (5)	RATIO (6)
	All reported occupations		99	100	96		
1	Professionals, etc.	151	84	84	90	24/18	133
3	Managers, etc.	269	76	99	83	17/17	100
4	Clerical, etc.	97	86	99	82	14/14	100
5	Sales	152	103	108	97	22/23	96
6	Craftsmen, etc.	567	120	111	95	92/82	112
7	Operatives, etc.	278	105	110	93	36/47	77
8-9	Service workers	202	110	99	109	44/55	80
11	Laborers (not farm or mine)	203	119	98	142	33/32	103
2	Farmers	111	94	73	93	11/6	183
10	Farm laborers	61	54	93	90	4/10	40

*Predicted Standardized Mortality Ratios are based on the occupation group differences in the proportions of men smoking various numbers of cigarettes, from Haenszel and associates,[11] and on the model of relative risks of lung cancer assigned to each smoking class, from Haenszel and Shimkin.[12]

†From Buechley and co-workers,[4] who provide crude estimates.

‡Number on death certificate divided by number according to occupational history. Histories were obtained by interview with hospitalized lung cancer patients who were subsequently traced on routine state death records.

toolmakers, meat cutters, and cooks vary significantly from the S.M.R.s for their respective social classes.

Lung cancer: Cancer of the trachea, bronchus, and lung increases slightly with lower social class of California men, aged 20 to 64, a trend which British mortality data revealed for the first time in 1949 to 1953. Incidence data by income class from the ten United States cities, 1947, for men of all ages, are consistent in direction, with the association appearing even more pronounced in magnitude.

Haenszel and co-workers[11] have reported some differences in current cigarette use between broad occupational groups. The proportion of cigarette smokers increases somewhat with lower social class, though heavy smoking does not increase in the same way. Since the association of cigarette smoking with lung cancer is quite strong, part or all of the social class variations in lung cancer mortality may be due to these smoking differences. The only available smoking data to test this are the national data reported by Haenszel and his co-workers showing 8 major occupation groups which correspond roughly with social class and which can be identified in the California occupation data.

Table XI carries the results of the test. The S.M.R.s that would be predicted from differences in cigarette smoking are computed from the proportionate distribution by current smoking amount in each major occupation group, and in the whole group, weighted by the relative risks of lung cancer assigned to each such smoking class as adapted by Haenszel and Shimkin[12] from prospective and

retrospective studies of lung cancer. It is not possible to adjust for age the smoking differences in occupational groups, but the refinement does not seem necessary since Haenszel and associates[11] report that the adjusted rates differ very slightly, if at all, from the crude rates for occupations.

There may be some error in the actual S.M.R.s because of misreporting according to major occupation group. Reproduced in column 6 of Table XI are crude estimates of the effect of that error as determined by Buechley and coworkers.[4] Column 2 gives the actual S.M.R.s due to lung cancer and column 3 the predicted (smoking) S.M.R.s.

Salesmen, craftsmen, and operatives have high predicted S.M.R.s with which their actual S.M.R.s tend to agree, but important divergences from the predicted S.M.R.s appear for the other occupational groups. Smoking practices would give service workers and laborers about the same predicted lung cancer experience as managers and clerical workers, yet the former have a risk of lung cancer death higher than that predicted from smoking, while the latter have a risk more favorable than expected according to their smoking. Some qualifications must be held in mind. The actual excess risk for service workers is due to the high rate for cooks only, though the occupation reporting bias estimates in column 6 suggest an underreporting of deaths for all service workers. Further, the actual S.M.R. for clerical workers is based on small numbers and does not differ significantly from the predicted S.M.R. On the other hand, the professionals are probably like the managers in having less lung cancer than expected according to smoking, since the bias estimate suggests an overreporting of deaths. The laborers, then, and possibly service workers, have more lung cancer deaths, and the managers, and possibly professionals, have fewer lung cancer deaths than one would predict from their use of cigarettes.

While the results are consistent with some social class difference in lung cancer death rates, they may also be open to explanation on other grounds. For example, the actual S.M.R.s are from California data while predicted S.M.R.s are based on national data, and we have no means of checking on the assumption that occupational makeup and smoking habits of broad occupational groups do not differ regionally. Or, again, the number of cigarettes smoked may not be an accurate measure of the amount of smoke presumably affecting the lungs. Laborers, for example, may leave a much smaller cigarette butt than managers (a practice which does seem to distinguish the British and American smokers[13] and might explain the higher rates among the former). But even if the latter point could explain the modest social class differences we have found in lung cancer, it may be somewhat more difficult to explain in the same way all of the stronger association found in the Ten City Incidence Survey.

The Census Intermediate Occupational List, used here, is hardly detailed enough for a satisfactory study of industrial risks of lung cancer, and we show the available data on specific occupations for whatever limited purpose they may be found to have. Among the 106 specific occupations, 12 showed significantly more deaths from lung cancer than expected according to the over-all rate. An additional 8 occupations had significantly fewer lung cancer deaths than expected.

VOL. 12 1960

The 20 occupations shown in Table XII are more than we are entitled to on the basis of sampling variation alone.

Some of the 20 occupations may vary from the average lung cancer mortality experience because of occupation reporting errors. Partly for this reason, and partly for the added knowledge it affords, Table XII also shows the S.M.R.s for all causes combined. Where there is a suspicion of a strong tendency to overreport or underreport the deaths in an occupation, the ratio of the lung cancer S.M.R. to the all causes S.M.R. may help to indicate whether there is an actual excess of lung cancer deaths. Some occupations with a high risk of lung cancer mortality have an equally high risk from all causes. Both relative risks for cooks

TABLE XII. MORTALITY FROM LUNG CANCER AND ALL CAUSES. SPECIFIC OCCUPATIONS WITH SIGNIFICANTLY DIVERGENT S.M.R.s FOR LUNG CANCER IN CALIFORNIA MEN, AGES 20 TO 64, 1949 TO 1951

SOCIAL CLASS	OCCUPATION	LUNG CANCER DEATHS			ALL CAUSES S.M.R.
		OBSERVED	EXPECTED	S.M.R.	
Lung Cancer S.M.R. Above 100					
I	Authors, etc.	11	6.3	175	107
III	Bookkeepers	12	6.4	188	100
III	Carpenters	108	74.7	145	89
III	Machinists, etc.	44	32.1	137	122
III	Plumbers, pipefitters	27	18.5	146	110
III	Stationary engineers	29	15.4	188	145
IV	Mine workers	29	11.6	250	225
V	Sailors, deck hands	11	3.8	289	272
IV	Taxicab drivers	16	7.4	216	139
IV	Cooks (not private)	44	24.4	180	184
V	Fishermen	11	5.6	196	106
V	Laborers (not farm or mine)	155	118.7	131	162
Lung Cancer S.M.R. Below 100					
I	College faculties	1	4.8	21	30
I	Civil engineers	4	10.9	37	89
I	Electrical engineers	1	5.8	17	72
II	Teachers (n.e.c.)*	4	12.5	32	64
II	Managers, trade	92	140.5	65	82
III	Other clerical workers	80	98.7	81	82
V	Janitors	33	47.1	70	78
V	Other specified laborers (mainly gardeners)	16	31.5	51	64

*N.e.c., not elsewhere classified.

approach twofold, and those for mine workers and sailors are greater. Consequently, we cannot say whether these occupations actually experience an excess risk of lung cancer, but if they do it is matched by an equally high general mortality liability.

Other occupations reveal considerably higher mortality ratios for lung cancer than for all causes: authors and journalists, bookkeepers, taxi drivers, and fishermen, each based on small numbers of lung cancer deaths; carpenters, plumbers

TABLE XIII. BRAIN CANCER CASES IN THE CALIFORNIA TUMOR REGISTRY BY AGREEMENT WITH PRIMARY CAUSE OF DEATH ON DEATH CERTIFICATES FOR EACH SOCIAL CLASS OF MEN, AGES 20 TO 64, 1942 TO 1958

	1942 TO 1947					1948 TO 1953					1954 TO 1958				
	DEATHS OF BRAIN CANCER CASES IN REGISTRY	DEATH CERTIFICATE DIAGNOSIS				DEATHS OF BRAIN CANCER CASES IN REGISTRY	DEATH CERTIFICATE DIAGNOSIS				DEATHS OF BRAIN CANCER CASES IN REGISTRY	DEATH CERTIFICATE DIAGNOSIS			
		SAME AS REGISTRY	UNSPECIFIED BRAIN TUMOR	BENIGN BRAIN TUMOR	OTHER CAUSE OF DEATH		SAME AS REGISTRY	UNSPECIFIED BRAIN TUMOR	BENIGN BRAIN TUMOR	OTHER CAUSE OF DEATH		SAME AS REGISTRY	UNSPECIFIED BRAIN TUMOR	BENIGN BRAIN TUMOR	OTHER CAUSE OF DEATH
	Number														
Total	207	147	29	15	16	276	219	26	11	20	310	250	22	9	29
Social Class I	15	14	1	0	0	26	22	3	0	1	19	15	2	0	2
Social Class II	44	35	4	2	3	67	56	5	0	6	68	52	5	3	8
Social Class III	63	39	11	7	6	95	77	7	5	6	119	99	8	0	12
Social Class IV	48	32	10	3	3	47	38	3	2	4	54	44	3	2	5
Social Class V	17	11	1	3	2	26	16	5	2	3	19	15	2	1	1
Farmers	11	8	2	0	1	6	3	3	0	0	19	18	0	1	0
Farm laborers	4	4	0	0	0	4	3	0	1	0	7	4	1	1	1
Occupation not reported	5	4	0	0	1	5	4	0	1	0	5	3	1	1	0
	Per Cent														
		SAME DIAGNOSIS	OTHER DIAGNOSIS				SAME DIAGNOSIS	OTHER DIAGNOSIS				SAME DIAGNOSIS	OTHER DIAGNOSIS		
Total	100	71	29			100	79	21			100	81	19		
Social Class I	100	93	7			100	85	15			100	79	21		
Social Class II	100	80	20			100	84	16			100	76	24		
Social Class III	100	62	38			100	81	19			100	83	17		
Social Class IV	100	67	33			100	81	19			100	81	19		
Social Class V	100	65	35			100	62	38			100	79	21		

VOL. 12 1960

and pipefitters, and stationary engineers, each based on somewhat larger numbers of lung cancer deaths. In so far as there are counterparts to these occupations in England and Wales, 3 showed significant or nearly significant excesses of lung cancer mortality in the latter area during 1949 to 1953[1]: fishermen, plumbers, gas workers, pipefitters, and drivers of passenger vehicles other than bus or coach. With the exception of fishermen, all showed somewhat less than average general mortality.

While these occupations might be considered suspect occupations until further knowledge sustains or disposes of that status, it is not easy to postulate a causative mechanism. A problem is also presented by the fact that some occupations restrict the opportunity to smoke cigarettes, while some may actually induce heavier smoking.

Brain and central nervous system: This site of cancer alone showed a direct association with higher social class that could not be explained by occupation reporting bias. At ages 20 to 64 the gradient increases from an S.M.R. of 58 for Social Class V to an S.M.R. of 127 for Social Class II, and, as noted earlier, the net occupation reporting error should not affect the S.M.R.s in these two classes. The British mortality data show an irregular gradient in the same direction at ages 20 to 64, and a stronger, more regular gradient at ages 65 and over.[1] But the Ten City Survey incidence data are inconsistent, showing almost the same liability to brain cancer at the highest and lowest income levels.

Such an inconsistency between mortality and incidence data could result if deaths among brain cancer cases in Social Class V were more frequently attributed to some primary cause other than brain cancer. We do know, from the report by Dorn and Cutler on the Ten City Survey,[2] that, compared to cases diagnosed as having cancer of other sites, a larger proportion of persons with an initial diagnosis of brain cancer will subsequently be recorded as having died from a cause other than malignancy. When the reports of newly diagnosed cases of cancer at all sites were compared with death certificates of each resident cancer patient reported to have died during the survey year or 6 months after, 12 per cent of death certificates recorded a different major site group and 6 per cent some primary cause of death other than cancer. For brain and central nervous system, a different major site group was recorded on 4 per cent, and a primary cause other than cancer on 40 per cent, of death certificates.

With data furnished by the California Tumor Registry,[14] we can see whether this known discrepancy between the case report of brain cancer and the subsequent assignment of cause of death is greater for Social Class V. The method is similar to that employed in discovering the discrepancy in the Ten City Survey except that hospital admission diagnoses of brain cancer found in the Registry records are checked against deaths occurring within 2 years after admission. Social class is assigned by coding the occupation entered on the death certificate.

Social Class V does in fact reveal, in Table XIII, bottom half, a greater amount of disagreement between diagnoses of brain cancer and the subsequent assignments of primary cause of death. During 1948 to 1953, which includes the period of the occupation-mortality study, only 62 per cent of deaths of the brain cancer cases in Social Class V were said to be due to a brain malignancy

compared to 85 per cent in Social Class I. The probability that this difference is due to chance is 5 per cent. The earlier period, 1942 to 1948, suggests the same social class difference. This is evidence that the social class gradient of mortality from brain cancer does not reflect the real incidence of the disease, for Social Class V had a greater incidence of brain cancer than the death records disclosed.

It was suspected by Dorn and Cutler[2] that the attribution of some other nonmalignant cause of death in such a large proportion of brain cancer cases was due to the inexact entry of "brain tumor" on the death certificate. Such entries, which fail to specify whether malignant or benign, are assigned to unspecified neoplasms and result in an undercount of brain cancer deaths. Table XIII, top half, indicates that this is not the only problem, for a large proportion of cases were certified on death to some cause of death other than brain tumor, whether malignant or benign.

An additional test of the proposition that mortality data show a spurious association between social class and brain cancer is suggested by the data of Table XIII. For all social classes combined there is a decrease from the year 1942 to 1958 in the proportion of benign and of unspecified brain tumors entered on death certificates. The proportion attributed to some cause other than brain tumor remains about the same, and the total amount of disagreement between case records and death records drops from 29 to 19 per cent. But more important, the social class differences in the amount of disagreement almost disappear by 1958. This trend suggests that another analysis of brain cancer mortality in 1960 should reveal no important social class differences if differential attribution of primary cause of death was responsible for the apparent mortality gradient in 1949 to 1951.

It is not clear why there is this time trend toward each social class showing approximately constant proportions of brain cancer cases being certified to some other primary cause at death. The trend is due not only to an increase of agreement between case and death records in Social Class V, but also to a decrease in such agreement for Social Class I from 1942 to 1958. There is some consistency in trend for Social Classes III, IV, and V over the period. The opposite trend for Social Class I is based on numbers too small for statistical significance, and yet it suggests that we do not know all the changes that may be taking place in diagnostic practices.

If the analysis of 1960 brain cancer mortality data shows the social class gradient persisting, contrary to our hypothesis that it should disappear, a different hypothesis suggests itself. This is, the incidence data of the Ten City Survey, showing no differential social class risk, could be in error if metastatic cancer was misdiagnosed as primary brain cancer to some important degree on hospital admission. This is a possibility because, while metastases from central nervous system cancers are rare, metastases to the central nervous system are not. According to Kernohan and Sayre[15] various attempts to estimate metastases to the brain range up to 40 per cent of all brain cancers. We do not, of course, know whether there is a large error in diagnosing metastatic cancer as brain cancer or whether such error, if it exists, is greater at lower socioeconomic levels. If true, the Ten City Survey incidence data which show no differences in social class risk

VOL. 12 196

may be misleading. At present, however, the apparent gradient of brain cancer mortality in California is the most likely candidate for disproof.

There were 5 occupations showing a significant excess of mortality from brain cancer, all of them white collar occupations: physicians in Social Class I; clergymen and designers, Class II; and "other" clerical workers and policemen, Class III. Significantly low S.M.R.s were found only for "other" service workers in Class IV, and janitors and laborers Class V.

Other sites: Where the California data failed to reveal gradients, that is, for pancreas, kidney, leukemia, and the lymphomas, the results are generally consistent with the Ten City Survey of incidence by income level. Not consistent, however, are the data from England and Wales, 1949 to 1953, showing moderate decreases with lower social level for cancer of the kidney, leukemia, and the lymphomas.

TABLE XIV. TOTAL MORTALITY AND MORTALITY EXCLUDING CERTAIN CAUSES. STANDARDIZED MORTALITY RATIOS BY SOCIAL CLASS FOR CALIFORNIA MEN, AGED 20 TO 64 DURING 1949 TO 1951

	ALL MEN WITH REPORTED OCCUPATIONS*	SOCIAL CLASS					FARMERS	FARM LABORERS
		I	II	III	IV	V		
All causes	96	87	85	94	98	132	93	90
All causes except violent causes†	96	92	88	97	98	121	90	78
All causes except violent causes and certain cancer sites‡	96	94	89	97	98	120	88	78

*S.M.R. for all men including unreported occupation is 100.
†Accidents, suicide, and homicide.
‡Cancer of upper alimentary tract, stomach, and lung.

Farmers and Farm Laborers.—Because these 2 occupations are subject to more serious occupation reporting bias, the results are discussed separately. One way to interpret the results is to compare the occupations on the basis of the ratio of the S.M.R. for each cancer site to the S.M.R. for all causes of death. This ratio, which should be unaffected by the reporting error, gives a picture only of the mortality from a specific cause relative to the mortality from all causes, on an age-standardized basis. The S.M.R.s for all causes were: farmers 93 (probably an inflated figure), farm laborers 90 (probably an underestimate). By comparing these figures with the S.M.R.s for specific cancer sites in Table III we can see that relatively few of the farm laborers' deaths were due to cancer. Since many farm laborers in California are migratory workers a greater proportion of their deaths may be due to infectious diseases and accidents. The highest cancer risks for farm laborers are caused by cancers of the esophagus and stomach, which, it should be noted, are also the highest cancer risks for farmers but not for men generally. The S.M.R.s for esophageal cancer are based on 23 deaths among

farmers and 14 among farm laborers, and for stomach cancer, 91 deaths among farmers and 61 among those they hire.

DISCUSSION

The laboring classes have a higher mortality risk resulting not only from the cancers mentioned here but also from diseases other than cancer. Table XIV illustrates this. There is an inverse association of social class with general mortality and also with general mortality excluding those cancer sites which also show the association. Line 2 of Table XIV, computed after deaths due to violent causes (accidents, suicides, and homicides) were subtracted from total mortality, indicates that the social class pattern is due to deaths following diseases (though the inverse association does not appear for coronary heart disease[3]). When it is remembered that in Social Classes I and IV the S.M.R.s are respectively raised and depressed by occupation misreporting, the gradient is actually more pronounced than Table XIV shows.

It is not clear what significance the higher general mortality risk has for the social class differences in cancer mortality. While it is possible that lower socioeconomic status carries exposure to a higher risk for a number of diseases, the reverse may also be true, that is, disease may increase the chances of a lowering of status or be an obstacle to increasing status. In the case of cancer, however, the short clinical history of most cancers may make it reasonable to dispose of the hypothesis that disease causes the status. For other diseases the hypothesis is not so readily dropped.

A general predisposition to disease, if it is more prevalent in laboring classes, conceivably could account for the social class gradients of both cancer and general mortality. That laboring men do not show a higher relative risk for all cancer, but only for sites comprising 39 per cent of total cancer mortality, may motivate research in environmental causes but does not discount entirely the predisposition hypothesis. A difference in general predisposition could produce inverse social class gradients, reinforce inverse gradients due to environmental causes, and even obscure gradients that would otherwise disclose direct associations with higher social class. These are only speculations, but, for many diseases, little is known about why social class gradients appear or, for that matter, do not appear.

SUMMARY

The social class risks of death from cancer among California men, at ages 20 to 64 in 1949 to 1951, were analyzed and compared with similar mortality experienced in England and Wales. Historical data on social class mortality are available for England but not for California.

As recently as 1930 to 1932 the mortality risk due to oral and pharyngeal cancer was greater in lower social classes in England and Wales. By 1950 the over-all death rate for these cancers, regardless of social class, had decreased considerably more in England and Wales than in the United States. The decrease was accompanied by the virtual disappearance of the social class gradient in the

VOL. 12 196

former country. California data, however, disclosed a pronounced social class difference in 1950.

Cancer of the stomach in England and Wales continued to show the same result from 1921 to 1951, that is, a twofold higher relative risk in lower social classes. In California the relative risk was about threefold in 1950. The over-all death rates for this site of cancer showed little or no change from 1930 to 1950 in the former country, but a 40 per cent decrease in the United States.

A similar social class risk for cancer of the esophagus seems to be changing in England and Wales, but was pronounced in California.

With the inclusion of cancer of the lung, which reveals a minor social class difference, lower social class is associated with an unfavorable relative risk of cancer at sites comprising 39 per cent of total cancer mortality in California men. Cancers of some other parts of the body show direct associations with higher social class but each requires additional evidence for confirmation.

OL.
12
960

REFERENCES

1. Registrar-General's Decennial Supplement, Occupational Mortality Part II, Vols. 1 and 2, London, 1958, H. M. Stationery Office.
2. Dorn, H. F., and Cutler, S. J.: Morbidity From Cancer in the United States. Public Health Monograph No. 56, Washington, D.C., 1959, U.S. Government Printing Office.
3. Breslow, L., and Buell, P.: Mortality From Coronary Heart Disease and Physical Activity of Work in California, J. CHRON. DIS. **11**:421, 1960.
4. Buechley, R., Dunn, J. E., Jr., Linden, G., and Breslow, L.: Death Certificate Statement of Occupation: Its Usefulness in Comparing Mortalities, Pub. Health Rep. **71**:1105, 1956.
5. Case, R. A. M.: Cohort Analysis of Cancer Mortality in England and Wales, 1911-1954 by Site and Sex, Brit. J. Prev. & Social Med. **10**:172, 1956.
6. Logan, W. P. D.: Occupational Mortality, Proc. Roy. Soc. Med. **52**:463, 1959.
7. Logan, W. P. D.: Personal communication.
8. Cutler, S. J., Schneiderman, M. A., and Greenhouse, S. W.: Some Statistical Considerations in the Study of Cancer in Industry, Am. J. Pub. Health **44**:1159, 1954.
9. Clemmesen, J., and Sorensen, J.: Carcinomas of the Digestive System in Denmark 1943-1956. (Cancer Incidence in Denmark, VI), Danish M. Bull. **6**:137, 1959.
10. Haenszel, W.: Variation in Incidence of and Mortality From Stomach Cancer With Particular Reference to the United States, J. Nat. Cancer Inst. **21**:213, 1958.
11. Haenszel, W., Shimkin, M. B., and Miller, H. P.: Tobacco Smoking Patterns in the United States. Public Health Monograph No. 45, Washington, D. C., 1956, U.S. Government Printing Office.
12. Haenszel, W., and Shimkin, M. B.: Smoking Patterns and Epidemiology of Lung Cancer in the United States: Are They Compatible? J. Nat. Cancer Inst. **16**:1417, 1956.
13. Doll, Richard: Smoking and Lung Cancer (A Report to the Sub-committee for the Study of the Risks of Cancer From Air Pollution and the Consumption of Tobacco), Acta internat. univ. contra cancrum **15**:1283, 1959.
14. Bragg, K., Linden, G., and Breslow, L.: The California Tumor Registry—a Summary Report on the Cancer Experience in 36 Hospitals. California Med. **87**:180, 1957.
15. Kernohan, J. W., and Sayre, G. P.: Tumors of the Central Nervous System; Section X, Fascicles 35 and 37, Atlas of Tumor Pathology Washington, D. C., 1952, Armed Forces Institute of Pathology.

LONG-TERM ANTICOAGULANT THERAPY IN AN OUTPATIENT DEPARTMENT. TECHNIQUES AND COMPLICATIONS

FRANK A. RIDDICK, JR., M.D.*
ST. LOUIS, MO.

From the Department of Medicine, Washington University School of Medicine, Barnes Hospital, and Washington University Clinics

(Received for publication March 29, 1960)

VOL. 12 1960

SINCE the early reports suggesting the benefits of long-term anticoagulant therapy in patients with myocardial infarction,[1-3] cerebral vascular disease,[4,5] and peripheral and cerebral thromboembolic phenomena,[6,7] interest in this form of therapy has continued to increase. In order to cope more effectively with the expanding use of anticoagulant therapy in patients at the Washington University Clinics and on the ward services at Barnes Hospital, an Anticoagulant Clinic, conducted and controlled by the House Staff, was established in 1956. The techniques of operation of this clinic have evolved gradually from the original plan; practical considerations and periodic reviews of the clinical experience have been responsible for the changes instituted.

The experiences of this clinic have been somewhat at variance with those mentioned in other published series, particularly with respect to the frequency of complications occurring during long-term anticoagulant therapy. We are prompted to report these observations since it is likely that they more closely reflect the experiences encountered in the private practice of medicine or in other large outpatient clinics than do those reported from investigational units concerned with the development of anticoagulant therapy.

MATERIAL AND METHODS

The Anticoagulant Clinic.—This clinic is a unit of the House Staff Medical Clinic of the Washington University Clinics. It is headed by the Assistant Resident who serves as admitting officer for the Ward Medical Service of Barnes Hospital for a 5 or 6 week period. The predominant sources of patients are the ward services of Barnes Hospital and the Washington University services at the St. Louis Veterans Administration Hospital. Anticoagulant therapy with bishydroxycoumarin (Dicumarol) is begun in the hospital. The ward physician

*Assistant in Medicine, Washington University School of Medicine, Chief Resident in Medicine, Barnes Hospital.

discusses the reasons for, details of, and dangers of long-term anticoagulant therapy with the patient. An attempt is made to select intelligent, reliable, cooperative patients who have an illness which might reasonably be expected to be benefited by anticoagulant treatment. Patients afflicted with neither severe hypertension nor a potentially hemorrhagic lesion, and who live close enough to permit weekly or biweekly visits are chosen.

The patient is followed in the Anticoagulant Clinic for regulation of the Dicumarol therapy, and in the Medicine or Neurology Clinic for the underlying disease. Periodic re-evaluations are made as to whether continued anticoagulant therapy is indicated. Most patients are seen at weekly intervals, although during times of bleeding and early in outpatient management, visits are more frequent. Many long-term patients with relatively stable prothrombin levels are seen at 2 or 3 week intervals.

On appointment days the Clinical Microscopy Laboratory determines the prothrombin time using the one-stage method of Quick[8] employing rabbit brain thromboplastin. The clinic physician informs the patient by telephone of the daily dose of Dicumarol and the time of the next visit. At intervals hemoglobin determinations, urinalyses, and stool guaiac analyses are performed. Clotting times are determined occasionally on patients who have been on therapy over 6 months.

Dicumarol is the only drug used; it was the first agent employed in the Clinic, and in our experience is somewhat less likely to give abrupt changes in prothombin concentrations between visits than faster acting anticoagulants. It has been expedient to use only one size tablet for therapy. Otherwise the chances for confusion in drugs and dosage would be very great since a new house officer rotates through the clinic every 5 weeks. The patients are largely uneducated and medically unsophisticated, and many patients receive their instructions by telephone during a busy clinic schedule.

Each patient is advised to report any bleeding episode immediately by telephone. When he is unable to contact the clinic, or if bleeding is brisk, he is directed to take one of the 5 mg. tablets of vitamin K_1 oxide (Mephyton) with which he was supplied and to come to the clinic or emergency room immediately. He is cautioned against ingestion of aspirin, salicylate-containing preparations, or vitamin capsules. Each patient is urged to discuss any proposed trip, or surgical or dental procedure with the clinic physician.

Control of Anticoagulant Therapy.—Prothrombin concentration of the patient's plasma has been maintained between 20 and 30 per cent of normal, corresponding to a prothrombin time of 30 to 23 seconds (the control in our laboratory is consistently 13.5 or 14 seconds). Forty per cent of normal (19.5 seconds) or greater has been considered as being well out of the therapeutic range, and 15 per cent of normal (> 40 seconds) or less has been considered as dangerously low. The prothrombin concentration was 40 per cent or more during 276 patient weeks of therapy and 15 per cent or less in 84 weeks; in 18 of the latter the level was below 10 per cent (> 56 seconds). Since the total number of patient-weeks was 4,800, the prothrombin concentrations were out of control 7.5 per cent of the time by the criteria adopted.

A variety of minor surgical and dental procedures have been performed on the patients. For most dental extractions and minor surgical procedures, the prothrombin concentration was allowed to rise to 50 to 60 per cent over a 1 to 2 week period. Anticoagulant treatment has usually been discontinued temporarily for major operative procedures. The preferred plan has been to allow a 2 week tapering off period preoperatively, with reinstitution of therapy as soon as possible in the postoperative course. Not all patients have had therapy withdrawn for operation; one of the patients successfully underwent ligation of the inferior vena cava while on full anticoagulant dosage.

Preparation for an emergency operation or therapy during a bleeding episode associated with a low prothrombin concentration has been managed by intravenous administration of small doses (less than 5 mg.) of vitamin K_1 oxide, rather than 50 mg. doses. In our experience the larger doses of vitamin K_1 oxide carry the hazard of precipitating a thrombotic episode and of making the patient relatively refractory to Dicumarol for several days. We have observed that during bleeding episodes as little as 0.5 mg. of vitamin K_1 oxide intravenously may raise the prothrombin concentration from less than 10 per cent to 20 or 30 per cent, and that bleeding may then stop.

When discontinuing anticoagulant therapy we again prefer a slow withdrawal of Dicumarol over a 2 to 4 week period.

One hundred and twenty-five patients have been followed in the Anticoagulant Clinic for periods ranging from 1 week to 32 months. The various categories of illnesses for which long-term anticoagulant therapy was given and the number of patients in each category are shown in Table I. The majority of patients in the coronary artery disease group were white men, and they account for the preponderance of white men in the entire series. The great majority of patients in the embolism group were women; no explanation for this predominance is readily apparent. Ages in the cerebral vascular disease group ranged from 39 to 82 years, with a mean age of 61 years. The patients with coronary artery disease were between the ages of 26 and 77 years, with a mean age of 52 years. In the group with thromboembolism, ages ranged from 34 to 76 years, the mean being 53 years. The patients have been followed on therapy for a total of 1,115 months and 19 days at the time of this survey, an average of 8 months, 24 days.

RESULTS

Of the 125 patients in the series, 66 remain on therapy. Sixteen died while on treatment; 10 were referred to physicians closer to their homes for continuation of anticoagulant therapy. Therapy was discontinued on the remaining 33 for a variety of reasons. These include: development of relative or absolute contraindication to continued anticoagulant therapy, such as carcinoma, peptic ulcer, or major operation (9 cases); unreliability or lack of cooperation (8 cases); short-term therapy indicated (8 cases); trial without therapy (6 cases); hemorrhage (1 case); and lack of sufficient indication on re-evaluation (1 case).

Of the group of 10 patients referred to other physicians, 4 have continued treatment for a period of 5 to 32 months. One died of congestive heart failure

VOL. 12 1960

TABLE I. CONDITIONS FOR WHICH OUTPATIENT ANTICOAGULANT THERAPY WAS EMPLOYED

DISEASE	NUMBER OF PATIENTS				
	TOTAL	MALES		FEMALES	
		WHITE	NEGRO	WHITE	NEGRO
Coronary artery disease	44	24	12	4	4
One myocardial infarction	26	11	11	2	2
Two or more myocardial infarctions	7	5	1	1	—
Angina pectoris or coronary insufficiency	10	8	—	—	2
Noncoronary chest pain	1	—	—	1	—
Cerebral vascular disease	55	19	13	9	14
Focal cerebral ischemic attacks without infarction					
Basilar-vertebral system	22	5	5	6	6
Carotid-middle cerebral system	19	11	1	2	5
Focal cerebral ischemic attacks with past infarction					
Basilar-vertebral system	6	2	3	—	1
Carotid-middle cerebral system	7	—	4	1	2
Cerebral infarction	1	1	—	—	—
Embolic disease	24	3	2	12	7
Rheumatic heart disease					
Cerebral or peripheral arterial emboli	4	1	—	2	1
Pulmonary emboli	3	—	—	2	1
Cerebral and pulmonary emboli	2	—	—	2	—
Nonrheumatic					
Renal artery embolus (?)	1	—	1	—	—
Pulmonary emboli					
Thrombophlebitis	4	—	1	—	3
Congestive heart failure	3	1	—	2	—
Carcinoma and operation	3	1	—	2	—
Pregnancy and delivery	1	—	—	—	1
No known predisposition	3	—	—	2	1
Miscellaneous	2	2	—	—	—
Thrombophlebitis without embolism	1	1	—	—	—
Primary pulmonary artery thrombosis	1	1	—	—	—
Total	125	48	27	25	25

4 months after leaving the clinic. Four others have stopped treatment 1 to 5 months later, 1 on his own advice, 1 on his physician's advice, and 2 on our advice (these patients were not being treated as recommended). No follow-up information is available on one.

Among the 33 patients in whom therapy was discontinued, 3 have died (1 from carcinoma of the lung 3 months after stopping therapy, another from congestive failure 7 months after stopping therapy, and the third from an unknown cause 5 months after anticoagulants were discontinued). Twenty-four patients continue to attend our clinics, follow-up periods range from 1½ to 26 months. None of this group has had a thromboembolic episode observed since therapy was stopped. The remaining 6 patients have been lost to follow-up; no complications were observed for 1 to 26 months after discontinuance of the therapy.

TABLE II. COMPLICATIONS OCCURRING DURING ANTICOAGULANT THERAPY

DISEASE	NO. OF PATIENTS	MEAN AGE	NO. OF TOTAL MONTHS OF THERAPY	AVERAGE NO. OF MONTHS	HEMORRHAGE						THROMBOSIS					OTHER DEATHS
					PATIENTS	MINOR	MAJOR	FATAL	HEMORRHAGE RATE PER 100 PATIENT-MONTHS	FATALITY RATE PER 100 PATIENT-MONTHS	PATIENT-	NON-FATAL EPI-SODES	FATAL EPI-SODES	THROMBOSIS RATE PER 100 PATIENT-MONTHS	FATALITY RATE PER 100 PATIENT-MONTHS	
Cerebral vascular disease	55	61 (39–82)	621	11.2	26	20	3	6	4.7	0.96	2	2	0	0.32	0	3
Coronary artery disease	44	52 (26–77)	364.5	8.3	12	17	2	1	5.5	0.27	4	3	1	1.1	0.27	1
Embolic disease	24	53 (34–76)	129.1	5.3	7	6	1	1	6.2	0.78	4	3	2	3.9	1.5	1
Other	2	52	1.1	0.6	0						0					0
Total	125	57 (26–82)	1,115.7	8.8	44	43	6	8	5.1	0.72	10	8	3	0.99	0.27	5
Patients on therapy longer than two months	88	56 (26–77)	1,079	12.2	33	38	5	3	4.3	0.28	6	7	1	0.75	0.09	4

VOL. 12 1960

Since we have no control series, comparison of our patients with untreated patients reported upon by others would not be of value in determining any benefit of therapy. It is of interest that none of our 44 patients on therapy because of coronary artery disease has suffered a detectable coronary thrombosis in 30.4 patient-years of therapy. One such episode occurred in a patient (Case 56) who had almost certainly discontinued therapy. One patient died with subintimal hemorrhage, and a patient with aortic stenosis died suddenly, no coronary occlusion or myocardial infarction being detected at autopsy. Probable myocardial infarction, characterized by chest pain, S-T segment depression, and transaminase rise, but without development of Q waves in the electrocardiogram, occurred in one patient in the cerebral artery disease group.

COMPLICATIONS OCCURRING DURING THERAPY

Table II depicts the number of complications encountered in each diagnostic group, in the entire series, and in the group of patients who were treated longer than 2 months.

1. Thrombosis.—Ten patients suffered thromboembolism while on anticoagulant therapy. Data on each of these cases are presented in Table III. In 5 of the patients, thrombosis or embolism occurred during the first 2 months of therapy.

An increased frequency of thromboembolism in the period of time immediately following discontinuation of anticoagulant therapy has been reported.[1,9-11] Thrombosis or embolism occurred during brief lapses of therapy in Cases 8,20, 54, and 56. In each case Dicumarol had been withdrawn by the physician or stopped by the patient abruptly, with no tapering off period. The terminal emboli in Case 8 and the carotid artery thrombosis in Case 71 followed administration of vitamin K_1 oxide.

Six of the thromboembolic episodes occurred while the patient's prothrombin concentration was within the therapeutic range. One occurred while the concentration was slightly above the upper limit of the range. The prothrombin concentration was normal at the time of the remaining 6 episodes, which followed abrupt withdrawal of Dicumarol or administration of vitamin K_1 oxide by 4 hours to 7 days.

2. Hemorrhage.—Thirty-six patients had a total of 57 hemorrhagic episodes. The largest number was in a 77-year-old woman who had 4 episodes of marked bruising after relatively insignificant trauma and one episode of bruising accompanied by hemarthrosis of the elbow joint.

The nature of these hemorrhagic episodes and their relation to inciting factors or to an abnormally low prothrombin concentration are shown in Table IV. It will be noted that approximately one-half the hemorrhagic episodes were related to the presence of either trauma or an underlying disease. In only 8 instances was bleeding associated with an abnormally low prothrombin level. Though the bleeding in the latter cases probably was related to the marked depression of prothrombin, several of our patients had prothrombin times of 1 to 2 minutes (< 5 per cent) without bleeding. One patient was blown through the wall of his

TABLE III. THROMBOSIS OCCURRING DURING THERAPY

CASE	AGE	RACE	SEX	DIAGNOSIS	OTHER DIAGNOSES	COMPLICATION	LENGTH OF OUTPATIENT TREATMENT AT TIME	PROTHROMBIN CONCENTRATION (%)	VITAMIN K_1 OXIDE	OUTCOME	REMARKS
Thrombosis in first 2 months											
20	68	W	M	Basilar artery insufficiency	Hypertensive cardiovascular disease	Left middle cerebral artery thrombosis	1½ mo.	70	—	Limited activity. Follow-up 23 months. Not on Dicumarol treatment	Had not taken Dicumarol for 4 days because of hematuria
56	70	W	M	Acute myocardial infarction		Myocardial infarction, multiple pulmonary emboli	12 days	100	—	Fatal. Autopsy performed	Missed appointment. Unlikely that he took Dicumarol
59	55	W	F	Multiple pulmonary emboli. Thrombophlebitis	Bronchiolar carcinoma with metastases	Pulmonary embolism	4 days	30	—	Fatal. Autopsy performed	
67	48	N	F	Pulmonary embolism	Hypertensive cardiovascular disease	Pulmonary embolism	1½ mo.	22	—	Inferior vena cava ligation. On treatment 3 months after ligation	
115	55	N	M	Myocardial infarction	Hypertensive cardiovascular disease	Left middle cerebral artery thrombosis	6 days	30	—	No residua. Not on treatment. Follow-up 2 months	
Thrombosis after 2 months											
8	67	W	F	Pulmonary and femoral arterial embolism	Arteriosclerotic heart disease. Chronic atrial fibrillation	1. Cerebral embolus	4 mo.	100	—		1. Had not taken Dicumarol for 5 days
						2. Pulmonary embolus	15 mo.	20	—		
						3. Cerebral and brachial artery embolism	15¼ mo.	100	50 mg. intravenous	Fatal.	3. Terminal emboli 4 days after ill-advised vitamin K_1 oxide therapy

VOL. 12 1960

CIRCULATION
VOL. 12
DEC. 1960

27	55	W	M	Myocardial infarction		Carotid artery insufficiency	25 mo.	30	—	No more episodes. Continues on Dicumarol treatment. Follow-up 2 months	
43	68	W	F	Basilar artery insufficiency	Hypertensive vascular disease	Myocardial infarction	9 mo.	28	— —	Doing well. Continues on treatment. Follow-up 18 months	
48	61	W	M	Thrombophlebitis. Suspected pulmonary embolism	Carcinoma of prostate	Pulmonary embolism	6 mo.	40	—	Doing well. Continues on treatment. Follow-up 8 months	
54	43	N	M	Myocardial infarction		Cerebral thrombosis	8 mo.	100	—	Not on treatment. Recovering slowly after 4 months	Given incorrect dose of Dicumarol followed by rapid rise of prothrombin concentration to 100% and cerebral thrombosis
Thrombosis immediately after therapy was discontinued											
71	59	N	F	Left carotid artery insufficiency	Hypertensive vascular disease	Left carotid thrombosis	9 mo.	—	50 mg.	Not on treatment. Aphasia and hemiparesis persist. Follow-up 3 wk.	Entered hospital with hematoma of thigh after fall. Vit. K_1 oxide given. 4 hours later, thrombosis of carotid artery occurred, proved by cerebral arteriogram. Endarterectomy performed

home by the St. Louis tornado of February, 1959, at a time when his one stage prothrombin concentration was 25 per cent. He suffered only minor lacerations and did not bleed excessively. Bleeding in 2 of the patients may have been associated with deficiency of plasma thromboplastin component, although there is no proof other than that bleeding episodes occurred with prothrombin concentrations of 30 per cent as measured by one-stage testing and with glass clotting times of 27 and 29 minutes.

TABLE IV. SITE AND SEVERITY OF HEMORRHAGE OCCURRING DURING ANTICOAGULANT THERAPY

	TOTAL NUMBER OF EPISODES	NUMBER OF EPISODES ASSOCIATED WITH TRAUMA OR UNDERLYING DISEASE	NUMBER OF EPISODES ASSOCIATED WITH A PROTHROMBIN <15 PER CENT
Minor hemorrhage			
Oral bleeding	1		1
Bleeding post extraction	1	1	
Hemoptysis	2		
Purpura	2		
Ecchymoses	15	13	1
Blood in stools	3	2	
Hematuria	15		5
Vaginal bleeding	1		1
	43*	16	6*
Major hemorrhage			
Hematuria	1		1
Hemarthrosis	2	2	
Bleeding duodenal ulcer	1	1	
Bleeding after biopsy of rectal polyp	1	1	
Hematoma	1	1	
	6	5	1
Fatal hemorrhage			
Cerebral hemorrhage	6	5	1
Subintimal hemorrhage	1	1	
Ruptured abdominal aneurysm	1	1	
	8	7	1

*Two patients each bled from 2 sites in a single episode.

A hemorrhage was judged to be of major proportions (6 instances) if hospitalization, transfusion, or operation was required to relieve bleeding or replace blood lost or if therapy was discontinued because of the episode. Minor episodes (43 instances) were controlled in almost all instances by decreased dosage or withdrawal of Dicumarol for several days; in several cases oral vitamin K_1 oxide or small intravenous injections (0.5 to 2 mg.) of vitamin K_1 oxide were employed to elevate the prothrombin concentration.

Eight deaths were associated with hemorrhage, although in some it is difficult to separate hemorrhage caused by the underlying disease from that associated

with anticoagulant therapy. Information about these patients is given in Table V. Six of the 8 deaths were due to cerebral hemorrhage. The diagnosis of a hemorrhagic complication was established or confirmed at autopsy in 6 of the 8 fatal cases and is likely in the other 2. With the exception of Case 11, the prothrombin concentration in these patients was between 19 and 30 per cent at the time of hemorrhage. None of these patients had severe hypertension.

3. Other Deaths.—Five other deaths occurred in the patients under therapy and could not be classified as either hemorrhagic or thrombotic with any degree of assurance.

Sudden death occurred in 2 patients. It was unexpected and unexplained in one; the other was a man with severe calcific aortic stenosis and angina pectoris. At autopsy no recent thrombus or hemorrhage was present.

Cerebral vascular accident occurred in 2. One died at home after 24 months of therapy. The other died of pneumonia, 2 weeks after developing aphasia and hemiparesis with clear spinal fluid. Both of these patients had cerebral arterial insufficiency syndromes as well as past cerebral infarction.

The remaining patient had had intractable congestive heart failure from rheumatic heart disease for 2 years. A cerebral embolus was the occasion for initiation of therapy. She died at home; no autopsy was performed.

DISCUSSION

The percentage of patients in our series who have had bleeding (35 per cent) is comparable to that of many reported series of treated outpatients. Nichol and Borg[12] reported bleeding in 36 per cent of 78 patients; Askey and Cherry[13] in 33 per cent of 20 patients; Bjerkelund[14] in 31 per cent of 119 patients; Fuller[15] in 48 per cent of 217 patients; Keyes[9] in 42 per cent of 121 patients, and Foley[16] in 36 per cent of 85 patients. In a deliberate search for bleeding with the use of orthotolidine testing of the urine, Peyman[17] found hemorrhage in 24 per cent of 34 outpatients. Other authors[1,2,18-22] have reported the incidence of hemorrhage in their series as 14 to 25 per cent. Manchester[23] reports that only 6 (2.9 per cent) of his 204 patients have bled in 1,064 patient-years of therapy.

The rate of bleeding, 5.1 episodes per 100 patient months, in our patients is somewhat higher than in most series. Most series have a rate of hemorrhagic complications between 0.85 and 2 per hundred patient months.[14,16,24-26] Although the present rate of hemorrhage in patients treated by Wright's group at Cornell is lower, earlier reports from this group had rates of 3.2 and 4.1 per 100 patient-months.[5,18] Fuller[15] found 5.9 hemorrhages per 100 patient-months in his experience; in the series reported by Peyman[17] there were 4.7 hemorrhages per 100 patient-months. The rate of hemorrhage in Manchester's patients is by far the lowest reported, 0.049 per 100 patient-months. This represents 1 bleeding episode for each 170 years of therapy.[23]

Most of the series of outpatients treated with anticoagulants are reported with the intent of showing the value of therapy in a particular group. Often the series represents a portion of the physician's experience chosen on the basis of diagnosis, the meeting of certain diagnostic criteria, or as part of a controlled series. Uncooperative patients are frequently eliminated from these series; and

TABLE V. FATAL HEMORRHAGE

CASE	AGE	RACE	SEX	DIAGNOSIS	OTHER DIAGNOSES	BLOOD PRESSURE	LESION	DIAGNOSIS ESTABLISHED BY	LENGTH OF OUTPATIENT TREATMENT AT TIME	PROTHROMBIN (%)	REMARKS
3	49	W	M	Myocardial infarction		120/70	Subintimal hemorrhage, right coronary artery	Autopsy	2 months	21	Sudden death. Subintimal hemorrhage, right coronary artery near origin. Hematoma narrowed, but did not occlude artery
4	65	W	M	Left middle cerebral artery insufficiency	Rheumatic heart disease. Hypertensive vascular disease. Diabetes mellitus	170/90–200/100	Ruptured abdominal aneurysm	Autopsy	1 month	22	Abdominal pain for 1 week before sudden death
5	64	N	F	Basilar artery insufficiency	Cerebral vascular accident in past	140/80	Massive intracerebral and intraventricular hemorrhage	Autopsy	6 days	19	
6	64	N	F	Left middle cerebral artery insufficiency	Hypertensive vascular disease	150/80–170/90	Massive thalamic and intraventricular hemorrhage	Autopsy	5 months	25	
11	56	W	F	Multiple pulmonary emboli	Arteriosclerotic heart disease	130/80	Cerebral hemorrhage, skin and mucous membrane hemorrhage	Clinical evidence	5 days	< 5 (over 3 min.)	Missed appointment. Continued to take medication despite spontaneous hematomas, epistaxis, oral and vaginal bleeding. After 3 days of bleeding, became comatose and entered hospital, exhibited stiff neck, fixed pupils, bilateral extensor plantar response
75	76	W	M	Basilar artery insufficiency	Arteriosclerotic heart disease. Arteriosclerosis obliterans	130/80	Intracerebral and intraventricular hemorrhage	Autopsy	6 months	25	
112	67	W	F	Basilar artery insufficiency	Right middle cerebral artery thrombosis in past. Hypertensive vascular disease	130/80–160/100	Cerebral hemorrhage	Clinical evidence	8 months	30	Fatal cerebral vascular accident with gross blood in cerebrospinal fluid
120	64	N	M	Basilar artery insufficiency	Cerebral vascular accident in past. Hypertensive vascular disease	170/90–200/100	Intracerebral and intraventricular hemorrhage	Autopsy	1¾ months	30	Glass clotting time, 27 min.

VOL. 12 1960

there is frequently a minimum period of therapy required for inclusion in the series (2 months to 1 year). Restriction of our group to those on therapy for over 2 months does not significantly alter the number or rate of hemorrhages. It does, however, eliminate 5 of the 8 deaths associated with hemorrhage, 2 of the 3 fatal thromboses, and one of the 5 deaths from other or uncertain causes. In our series elimination of patients treated less than 2 months removes the group who are unreliable or uncooperative and those in whom distance or debility prevent carrying out the program. We suspect that it also tends to eliminate those patients who die as a result of the episode for which anticoagulant therapy was initiated and some who have an underlying disease which will become manifested by severe or fatal hemorrhage.

It is difficult to find the mortality rate due to anticoagulant therapy. This is more properly termed death associated with bleeding since in most cases hemorrhage due to underlying disease cannot be separated accurately from that caused by anticoagulants. Regardless of the initial cause of bleeding it is difficult to conceive of benefit accruing to a patient from being treated with an anticoagulant drug at a time when he starts bleeding, and is easy to visualize the hazard of such a situation. Several series contain from 1 to 6 deaths associated with hemorrhage.[9,12,14,18,19,21,22,24,26] These are large series and the percentage of patients dying of hemorrhage in them is 0.5 to 3 per cent, most being about 1 per cent or below.

The death of 1 of our patients (Case 3) occurred at the time of a hemorrhage into the base of an atherosclerotic plaque. Although some pathologists feel that subintimal hemorrhage is an important factor in initiation of the process of coronary occlusion and have described its presence in up to 89 per cent of cases of coronary occlusion,[27-30] the complication has not been regarded as a problem in anticoagulant therapy of acute myocardial infarction by some authorities[31,32]; nor has it been mentioned as a factor in myocardial infarction occurring in patients on long-term anticoagulant therapy. In 2 of Bjerkelund's 24 patients on whom autopsy was performed there were small hemorrhages in the base of atherosclerotic plaques.[14] In only one of these was the arterial lumen narrowed; both patients had died of cerebral hemorrhage. Special techniques are necessary for the systematic study of subintimal hemorrhages[33]; perhaps these techniques will shed some light on the frequency and importance of subintimal hemorrhage in anticoagulant therapy of coronary artery disease.

Cerebral hemorrhage was the cause of death in 6 patients in our series. One was almost certainly associated with an excessive dose of anticoagulant; the remaining 5 were all from the group with cerebral artery disease and had prothrombin times within the therapeutic range at the time of hemorrhage. Eleven patients in our series had cerebral vascular accidents or "strokes." Eight of these episodes were fatal; six were cerebral hemorrhages; the exact nature of the process is not known in the other two.

The most common cause of fatal bleeding in outpatients taking anticoagulants seems to be cerebral hemorrhage.[1,12,14,19,24,26] Most large series are of patients with coronary artery disease, and one might expect this complication more frequently in a population composed of individuals with symptomatic cerebral

arteriosclerosis. In patients with coronary artery disease Bjerkelund[14] found comparable numbers of cerebral hemorrhages occurring in patients treated with Dicumarol (4 of 179 patients) as in control patients (5 of 118 patients). The cerebral hemorrhage was fatal in all 4 patients on anticoagulants, but in only one of the 5 control patients.

In giving anticoagulants to 100 outpatients with cerebral vascular disease, the majority of whom had embolic disease, McDevitt and co-workers[26] discovered 5 cases of cerebral hemorrhage, 3 of which were fatal. This same group recently reported a series of 108 hospitalized patients with cerebral thrombosis, treated with anticoagulants. Forty-seven of this number had a vascular accident as a complication of myocardial infarction. There were 4 cerebral hemorrhages, of which 3 proved fatal,[34] although one death may not have been related to anticoagulation. Vastola and Frugh[35] administered both heparin and warfarin to 55 hospitalized patients with recent cerebral thrombosis. Four of this number died with massive cerebral hemorrhages. The authors concluded that the risk of cerebral hemorrhage was too great to warrant such therapy. Barron and Ferguson[36] were able to discover in the literature 58 instances of intracranial bleeding in association with anticoagulant therapy; to these they added 4 cases of their own. Almost one half of the patients included in their report had subacute bacterial endocarditis; the remaining 35 cases would not appear to be an impressive number in view of the immense numbers of both hospitalized and ambulatory patients given heparin or prothrombin depressant drugs.

Some investigators have suggested that at least some bleeding in dogs given Dicumarol is on the basis of Dicumarol-induced vascular lesions.[37] In humans, hemorrhagic necrosis of the skin has been attributed to injury of the capillaries by Dicumarol,[38] but biopsy was not performed in the cases reported.

It is more likely that the lethal potentialities of Dicumarol in patients with cerebral vascular accidents lie in the danger of converting a small hemorrhage to a large one or of transforming ischemic infarction of the brain into hemorrhagic infarction. Animal experiments tend to confirm this. Whisnant[39] has shown that in dogs treated with Dicumarol and Tromexan, infarction of the brain by intracarotid injection of liquid vinyl acetate tends to be hemorrhagic in nature, while this agent causes ischemic infarction in the absence of anticoagulant treatment. Sibley and co-workers[40] created cerebral infarction in dogs by injection of preformed clot into the carotid artery. This technique frequently gives hemorrhagic infarction of the brain in normal dogs.[39] Of the immediate survivors, the number showing hemorrhagic infarction was no greater in the group given anticoagulants; however, the severity of hemorrhage and the number of deaths was greater than in the control group.

Although there is very little evidence to indicate that therapy with prothrombin depressant drugs in customary doses causes cerebral hemorrhage, it is likely that when a cerebral vascular accident occurs in a patient on such therapy he is in greater danger than he would be if not on therapy. The magnitude of this risk is not known but would seem greater in those patients who are selected for therapy because of symptoms of cerebral atherosclerosis or cerebral emboli than in patients with myocardial infarction or pulmonary emboli. This risk must be balanced against expected benefits of anticoagulant therapy.

VOL. 12 1960

Our series does not permit any conclusion about the benefits of long-term anticoagulant therapy. The paucity of coronary artery thrombosis (one probable case) is of interest, as is the fact that 6 of the 13 observed thromboembolic episodes followed a rapid return of the prothrombin concentration to normal levels.

Most of the series in the literature indicate that in certain conditions long-term anticoagulant therapy is beneficial in terms of decreasing recurrence of and mortality from thromboembolism. Most of these series lack rigid controls, however, and do not provide definite proof of benefit from anticoagulants.

In three studies of the value of anticoagulant therapy following myocardial infarction, careful controls have been used. None of these studies was performed as a double blind experiment. Bjerkelund[14] was able to demonstrate benefit from long-term Dicumarol administration after myocardial infarction only in patients of both sexes below the age of 60 years, in patients with their first myocardial infarction, and in the first year of therapy following infarction. In a study by a working party of the Medical Research Council rigid criteria were adopted to select 2 homogeneous groups, one of which received therapeutic doses of 2-phenylindanedione, while the other received doses of the drug so low as not to affect the prothrombin time. Three hundred and eighty patients from 15 hospitals were followed. In this study a significant difference from the low dosage group could be established in treated male patients, especially below the age of 55, and in patients with more than one infarction. In treated men, mortality was significantly lower during the first 3 months but not thereafter, although the reinfarction rate was lower than in the low dosage group throughout the 2 year study.[24] The authors of the report felt that it was not valid to compare data from the 2 groups existing after one year of therapy because of the inclusion in the high dosage group of patients who would have died had they not received treatment. McMichael[41] has criticized the separation of data from age and sex groups, to find a group significantly different from the controls.

In a study covering a 10-year period Manchester[23] amassed a series of 204 cooperative patients who continued anticoagulant therapy after a myocardial infarction. Twenty-nine had subsequent myocardial infarction, and 6 died as a result. In a control group of 200, which appears comparable to the treated group except for an incidence of congestive heart failure which was almost twice as great as in the treated group, 68 patients suffered myocardial infarction, from which 36 died.

There is evidence that mortality is lower in patients with severe angina pectoris, coronary insufficiency, and impending myocardial infarction treated with long-term anticoagulants than in similar patients reported in follow-up series not employing anticoagulants.[19,21] No series utilizing comparable control groups has yet been reported.

There is a similar lack of comparable control groups in studies of the value of anticoagulant therapy in preventing further systemic arterial embolization in rheumatic heart disease, although if the patients themselves are used as controls, many fewer emboli are seen when the patients are on anticoagulants than when they are not.[6,7,16,42]

In the field of cerebral vascular diseases Millikan[43] and his group at the Mayo Clinic have reported relief of attacks of intermittent insufficiency of the basilar-

vertebral system or carotid system with anticoagulant therapy. Eight per cent of 107 patients with vertebral-basilar thrombosis treated with anticoagulants died, while 58 per cent of 31 patients with this condition studied earlier died. Two of 31 treated patients with progressive thrombosis of the carotid artery developed hemiparesis; of 17 untreated patients studied earlier 5 became hemiparetic and 2 died. McDevitt[26] and the group from Cornell reported the results obtained from a study of 100 patients with thrombotic or embolic cerebral vascular disease treated with anticoagulation. The majority of this group had embolic phenomena rather than cerebral atherosclerosis. Many fewer episodes occurred in periods when these patients were being treated compared to periods when they were not. This technique of comparison tends to emphasize differences in results between the two periods of time, since, as this same group has shown, the rate of thromboembolism after discontinuance of anticoagulant therapy is somewhat higher than the rate after a thromboembolic episode without anticoagulant therapy.[10]

Fisher[44] felt that there was benefit from anticoagulant therapy in reduction of number of attacks of cerebral ischemia and number of strokes. This benefit could be detected in those who had transient attacks of cerebral ischemia, with or without moderate neurological deficit, and in those treated during the course of progression of a stroke when they were compared with a group of nonrandom untreated patients. No improvement could be detected in patients in whom the full-blown picture of cerebral infarction was present. In this series therapy was not continued longer than 6 months in most patients.

The difficulty in predicting the future course of a patient with transient episodes of cerebral ischemia, the risk of cerebral hemorrhage, and the difficulties encountered in long-term management of patients in the older age groups, some with neurological deficits, have caused investigators in this field to appreciate the need for a carefully controlled study with long-term follow-up. A preliminary report of such a study has been given by the Cornell group.[45] The number of patients is small at present, but a smaller number of thromboembolic episodes have occurred in the treated group. The type of thromboembolic episodes has not been given, and there is as yet no information as to whether cerebral thrombosis occurs less often with long-term anticoagulant treatment.

SUMMARY AND CONCLUSIONS

The details of establishing an anticoagulant clinic are given, and the results of its first 3 years of operation are presented and discussed. One hundred and twenty-five patients have been followed for 1,116 months on therapy for coronary artery disease, cerebral vascular disease, and pulmonary and systemic arterial embolism.

Forty-four patients experienced 57 bleeding episodes of which 6 were major and an additional 8 fatal. Cerebral hemorrhage accounted for 6 of these; subintimal hemorrhage and bleeding from abdominal aortic aneurysm, one each. The implications of cerebral hemorrhage during anticoagulant therapy are discussed. It is suggested that anticoagulant therapy has an adverse effect on cerebral infarction and cerebral hemorrhage in patients on long-term therapy.

Six thromboses occurred shortly after anticoagulant therapy was discontinued; in 2 of these large doses of vitamin K_1 oxide were administered. The implication of this is discussed.

The literature concerning beneficial results of long-term anticoagulant therapy is discussed, as is the need for more carefully controlled studies on coronary insufficiency, cerebral vascular disease, and peripheral embolization.

No conclusion can be reached from this series about the efficacy of anticoagulant therapy, although it is of interest that only one probable coronary thrombosis occurred in the series of 125 patients. None of the 44 patients on therapy because of coronary artery disease suffered coronary thrombosis.

OL.
12
960

REFERENCES

1. Nichol, E. S., Phillips, W. C., and Jenkins, V. E.: Anticoagulants in Coronary Heart Disease, M. Clin. North America **38**:399, 1954.
2. Owren, P. A.: Long Term Anticoagulant Therapy in Coronary Artery Disease, Schweiz. med. Wchnschr. **84**:822, 1954.
3. Keyes, J. W., Drake, E. H., James, T. N., and Smith, F. J.: Survival Rates After Myocardial Infarction With and Without Long Term Anticoagulant Therapy, Am. J. M. Sc. **226**:607, 1953.
4. Siekert, R. G., Millikan, C. H., and Shick, R. M.: Current Indications for Use of Anticoagulant Drugs in Cerebral Vascular Disease, Circulation **13**:725, 1956.
5. Wright, I. S., and McDevitt, E.: Cerebral Vascular Disease—Significance, Diagnosis, and Present Therapy, Lancet **2**:825, 1954.
6. Wood, J. C., and Conn, H. L.: Prevention of Systemic Arterial Thrombi in Chronic Rheumatic Heart Disease by Means of Protracted Anticoagulant Therapy, Circulation **10**:517, 1954.
7. Cosgriff, S. W.: Chronic Anticoagulant Therapy in Recurrent Embolism of Cardiac Origin, Ann. Int. Med. **38**:278, 1953.
8. Quick, A. J., Stanley-Brown, M., and Bancroft, F. W.: A Study of the Coagulation Defect in Hemophilia and Jaundice, Am. J. M. Sc. **190**:501, 1935.
9. Keyes, J. W., Drake, E. H., and Smith, F. J.: Survival Rates After Acute Myocardial Infarction With Long Term Anticoagulant Therapy, Circulation **14**:254, 1956.
10. Carter, S. A., McDevitt, E., Gatje, B. W., and Wright, I. S.: Analysis of Factors Affecting the Recurrence of Thromboembolism Off and On Anticoagulant Therapy, Am. J. Med. **25**:43, 1958.
11. Sise, H. S., Gauthier, J., and Becker, R.: Risk of Interrupting Long-Term Anticoagulant Treatment (abstract), Circulation **20**:771, 1959.
12. Nichol, E. S., and Borg, J. F.: Long Term Anticoagulant Therapy to Prevent Recurrent Coronary Artery Thrombosis, Circulation **1**:1097, 1950.
13. Askey, J. M., and Cherry, C. B.: Thromboembolism Associated With Auricular Fibrillation. Continuous Anticoagulant Therapy, J.A.M.A. **144**:97, 1950.
14. Bjerkelund, C. J.: The Effect of Long Term Treatment With Dicoumarol in Myocardial Infarction, Acta med. scandinav. Supp. **330**:1, 1957.
15. Fuller, J. A.: Experiences With Long Term Anticoagulant Therapy, Lancet **2**:489, 1959.
16. Foley, W. T., McDevitt, E., Symons, C., and Wright, I. S.: Further Experiences With Long Term Anticoagulant Therapy, A.M.A. Arch. Int. Med. **95**:497, 1955.
17. Peyman, M. A.: Significance of Hemorrhage During Therapy of Patients With the Coumarin Anticoagulants, Acta med. scandinav. Supp. **339**:1, 1958.
18. Tulloch, J., and Wright, I. S.: Long Term Anticoagulant Therapy—Further Experiences, Circulation **9**:823, 1954.
19. Waaler, B. A.: The Effect of Permanent Anticoagulant Therapy on Symptoms and Mortality in Angina Pectoris, Acta med. scandinav. **157**:289, 1957.
20. Grant, W. G., and McMillan, R. L.: Experience With Out-Patient Anticoagulation Therapy, Canad. M. A. J. **78**:317, 1958.
21. Nichol, E. S., Keyes, J. W., Borg, J. F., Coogan, T. J., Boehrer, J. J., Mullins, W. L., Scott, T., Page, R., Griffith, G. C., and Massie, E.: Long Term Anticoagulant Therapy in Coronary Atherosclerosis, Am. Heart J. 55:142, 1958.
22. Suzman, M. M., Ruskin, H. D., and Goldberg, B.: An Evaluation of the Effect of Continuous Long Term Anticoagulant Therapy on the Prognosis of Myocardial Infarction, Circulation **12**:338, 1955.
23. Manchester, B.: *a*. Prevention of Myocardial Infarction, A.M.A. Arch. Int. Med. **100**:959, 1957. *b*. The Value of Continuous Long Term Anticoagulant Therapy, Ann. Int. Med. **47**:1202, 1957.

24. Report of Working Party to Medical Research Council: An Assessment of Long Term Anticoagulant Administration After Cardiac Infarction, Brit. M. J. **1**:803, 1959.
25. Bay, E. B., Adams, W. R., Jones, R. J., Page, R. G., and Seide, M.: Anticoagulant Drugs in Ambulatory Patients, Circulation **9**:741, 1954.
26. McDevitt, E., Carter, S. A., Gatje, B. W., Foley, W. T., and Wright, I. S.: Use of Anticoagulants in Treatment of Cerebral Vascular Disease, J.A.M.A. **166**:592, 1958.
27. Paterson, J. C.: Capillary Rupture With Intimal Hemorrhage as a Causative Factor in Coronary Thrombosis, Arch. Path. **25**:474, 1938.
28. Paterson, J. C.: Some Factors in the Causation of Intimal Hemorrhages and in the Precipitation of Coronary Thrombi, Canad. M.A.J. **44**:114, 1941.
29. Wartman, W. B.: Occlusion of the Coronary Arteries by Hemorrhage Into Their Walls, Am. Heart J. **15**:459, 1938.
30. Horn, H., and Finkelstein, L. E.: Arteriosclerosis of the Coronary Arteries and the Mechanism of Their Occlusion, Am. Heart J. **19**:655, 1940.
31. Wright, I. S., Marple, C. D., and Beck, D. F.: Myocardial Infarction—Its Clinical Manifestations and Treatment With Anticoagulation, New York, 1954, Grune & Stratton, Inc.
32. Friedberg, C. K.: A Critical Appraisal of Anticoagulants for Short Term and Long Term Use in the Management of Myocardial Infarction and Systemic Arterial Embolism—Part I, New York J. Med. **58**:877, 1958.
33. Blake, T. M., and Springer, P. K.: Intramural Hemorrhage in Coronary Atherosclerosis, Circulation **17**:907, 1958.
34. Groch, S. N., Hurwitz, L. J., McDevitt, E., and Wright, I. S.: Problems of Anticoagulant Therapy in Cerebrovascular Disease, Neurology **9**:786, 1959.
35. Vastola, E. F., and Frugh, A.: Anticoagulants for Occlusive Cerebrovascular Disease, Neurology **9**:143, 1959.
36. Barron, K. D., and Ferguson, G.: Intracranial Hemorrhage as a Complication of Anticoagulant Therapy, Neurology **9**:447, 1959.
37. McCarter, J. C., Bingham, J. B., and Meyer, O. O.: Studies on the Hemorrhagic Agent 3,3′Methylenebis (4-Hydroxycoumarin). IV. The Pathologic Findings After the Administration of Dicumarol, Am. J. Path. **20**:651, 1944.
38. Jordal, R.: Necrosing Cutaneous Hemorrhages as a Complication in Dicumarol Treatment, Acta med. scandinav. **154**:477, 1956.
39. Whisnant, J. P.: Experimental Cerebral Vascular Disease and Dysfunction, *in* Wright, I. S., and Millikan, C. H.: Cerebral Vascular Disease, New York, 1958, Grune & Stratton, Inc., p. 53.
40. Sibley, W. A., Morledge, J. A., and Lapham, L. W.: Experimental Cerebral Infarction: The Effect of Dicumarol, Am. J. M. Sc. **234**:663, 1957.
41. McMichael, J.: Long Term Anticoagulant Therapy, Brit. M. J. **1**:970, 1959.
42. Owren, P. A.: The Technic of Permanent Anticoagulant Treatment, Postgrad. Med. **21**:83, 1957.
43. Millikan, C. H., Siekert, R. G., and Whisnant, J. P.: Anticoagulant Therapy in Cerebral Vascular Disease—Current Status, J.A.M.A. **166**:587, 1958.
44. Fisher, C. M.: Use of Anticoagulants in Cerebral Thrombosis, Neurology **8**:311, 1958.
45. McDevitt, E., Groch, S. N., and Wright, I. S.: Cooperative Study of Cerebrovascular Disease—Methodology and a Preliminary Report on the Use of Anticoagulants, Circulation **20**:215, 1959.

VOL 12 196

THE AGED RECLUSE—AN EXPLORATORY STUDY WITH PARTICULAR REFERENCE TO COMMUNITY RESPONSIBILITY

RUTH GRANICK, B.A., AND FREDERIC D. ZEMAN, M.D.
NEW YORK, N. Y.

From the Medical Services, The Home for Aged and Infirm Hebrews of New York, and the Department of Sociology, Columbia University, New York

(Received for publication March 31, 1960)

OL.
12
960

IN spite of the increasing attention paid to the problems of old age by social scientists and physicians, many fields of potential joint interest remain untouched. Among these may be mentioned criminality, alcoholism, and social isolation in the aged. The present study attempts to explore an extreme form of isolation exemplified by recluses, which has received more attention in the newspapers than it has in professional literature.

Perusal of *Psychological Abstracts* covering the period from 1927 to 1959 reveals no index headings for either the term "hermit" or "recluse." The relatively synonymous headings of "asceticism" or "isolation" do not deal with individuals called to mind by the term recluse. The *Psychological Abstracts* contain brief reviews of papers which appeared in about 450 journals published throughout the world in the field of psychology, psychiatry, sociology, anthropology, and related fields.

Despite the furor created in the press by the discovery of the junk-filled home in Harlem in which the Collyer brothers were found dead in 1947, there are no studies made of the Collyers by personnel trained either in the medical or behavioral sciences. The only volume devoted to this general topic is a collection of life histories of hermits and recluses written in a vivid and entertaining style by Helen Worden Erskine, whose interest as a newspaper reporter was first aroused by the Collyer case.[1]

What accounts for the general public interest in these stories? Apparently the lives of recluses contain the ingredients recognized as essential to a good newspaper "story." The people involved are deviants who are often surrounded by mystery and violence. Frequently they are possessors of unsuspected treasures. One can hardly deny the popular appeal of such recent headlines as "Recluse Leaves $150,000 to a Hospital in Danbury" or "Timken Widow, a Recluse, Dies Amid Art Treasures." However, fascinating as these stories of aberrant behavior are to the reader of newspapers, for some reason scientists seem to have been frightened off with the result that serious research on recluses is not to be found.

The purpose of this paper is, therefore, to explore in a relatively systematic manner some characteristics of aged behavioral deviants, and to stimulate not only interest but more careful studies.

DEFINITIONS

What is a recluse? According to Webster's New International Dictionary,[2] the term recluse refers to: 1. "A person who lives in seclusion, as a hermit* or monk; 2. One who leads a retired life with few social contacts." This definition is based solely on the overt behavior of the individual and does not distinguish between those who "retire" in a socially acceptable manner such as monks and ascetics, and those who "retire" for so-called pathologic reasons such as fear or mental illness. Nor does the dictionary's definition distinguish between individuals who withdraw of their own volition and those who are involuntarily secluded by reason of disorders associated with advancing years. The aged recluses to be discussed in this paper appear to retire for pathologic reasons though it is difficult to be certain of this in specific cases.

At this point it seems reasonable to ask if the subject of the aged recluse is worthy of any serious research effort. There are at least 3 reasons for answering this question in the affirmative: one is that any behavioral aberrations or deviations from normative expectations are interesting subjects for study in their own right; a second reason is that the factors which isolate aged recluses may be related to those which isolate the aged in general; and the third reason is that the presence of recluses in any community often raises difficult problems for the other residents.

METHOD

The data for this paper were furnished by one of the authors (F. D. Z.) who has been clipping newspaper articles on recluses out of metropolitan papers over the last 17 years, as part of his over-all concern with the problems of older people. The rather arbitrary criteria according to which "recluses" were selected for this study were as follows: 1. They were 56 years of age or older. 2. They were either labeled a recluse by the newspaper, or the label was implied by their mode of life.

Needless to say, this collection of cases does not meet the requirements of either an unbiased sample of recluses as described in the newspapers or a random sample of recluses in the population; thus the findings based on this group of cases must be regarded as tentative. Nevertheless, in the absence of detailed case histories of recluses, these available clippings give at least preliminary information which may serve to initiate study and stimulate further investigation. In all, 105 recluses comprise the sample. Data about them were collected from 7 metropolitan New York newspapers[3] covering the years of 1942 to 1959 but omitting 1943, 1944, 1945, 1948, and 1956.[4] These clippings usually contain scanty information and rarely refer either to background factors or other forces

*The word "hermit" is listed as a synonym for recluse with the following definition: "A person who retires from society especially from religious motives."

VOL. 12 196

which might influence an individual to retire from the world; nor do they ever contain information on the duration of the recluse's seclusion. Thus, the newspaper accounts represent a sample of elderly individuals who have lived secluded lives for an unknown quantity of time and for unknown motives and who, for one or another reason, are considered newsworthy.

In Table I are summarized the primary reasons for these individuals coming to the attention of the press. Four large groups may be identified in the 105 cases. Thirty-seven, or 35 per cent, aroused interest because they were discovered at home either dead or near death from apparently natural causes. In thirty-two cases, or 30.5 per cent, a large sum of money was discovered at death. Eleven cases, or 10.5 per cent, were involved in some form of violence, either in an accident or death under violent circumstances, often a criminal attack. Thus 80 cases, or 76.2 per cent, achieved newspaper notice at point of death. The remaining 25 cases, or 23.8 per cent, were brought to the attention of authorities by some form of eccentric or unusual behavior.

OL.
12
960

FINDINGS

What kind of people are recluses? In describing this sample the recluse's characteristics will be arranged under the following headings: 1. General attributes, 2. Social relationships, 3. Patterns of behavior, and 4. Final status.

1. General Attributes.—Of the total sample of 105 recluses aged 56 years and over, the largest proportion, 37 per cent, falls within the 76- to 85-year-old group. The next largest proportion, 25.7 per cent, are to be found in the 66 to 75 year range. Only 12 per cent fall within the 86 to 105 year-old group. Of possible significance as to early age of onset of causes leading to reclusiveness is the occurrence of 19 cases, or nearly 20 per cent, in the under 65 year class (Table II).

Study of sex incidence shows approximately the same proportion of male and female recluses, with about 50 per cent in each category.

Most of these recluses were located in New York City and other eastern cities. There were relatively few recluses in rural areas included in the series. Needless to say, this is greatly influenced by the fact that metropolitan papers were used as sources of data (Table III).

TABLE I. PRIMARY REASON FOR RECLUSE COMING TO THE ATTENTION OF THE PRESS

	FREQUENCIES	
	NUMBER	PER CENT
Discovered at home either dead or near death (from apparently natural causes)	37	35.2
Large sum of money discovered upon death	32	30.5
Brought to the attention of authorities by some form of "eccentric" or unusual behavior	25	23.8
Involved in some form of violence: e.g., in accident or died under violent circumstances	11	10.5
Total	105	100

TABLE II. AGE DISTRIBUTION OF AGED RECLUSES

	FREQUENCY DISTRIBUTION	
	NUMBER	PER CENT
56– 65	19	18.1
66– 75	27	25.7
76– 85	39	37.1
86– 95	11	10.5
96–105	2	1.9
Unspecified	7	6.7
Total	105	100

TABLE III. LOCATION OF RECLUSES BY REGION AND WHETHER RURAL OR URBAN

	FREQUENCY DISTRIBUTION	
	NUMBER	PER CENT
New York City and other eastern cities	70	66.7
Cities in west, far west, south and outside United States	20	19.0
Upper New York State and other rural and suburban areas	14	13.3
No information	1	1.0
Total	105	100

The recluses who lived in apartments numbered 43.8 per cent, while 28.6 per cent resided in their own homes, and another 10.5 per cent lived in hotel suites. Makeshift homes such as shacks, garages, or basements housed 6.7 per cent. On 10.5 per cent there was no information as to housing (Table IV).

Since no information on country of birth was available for 74 per cent of the cases, it can be mentioned only in passing that in the remaining cases, 15 per cent were born in the United States and 11 per cent in Europe.

About equal proportions had the following occupational histories: 18 per cent were in manual and service occupations, 18 per cent in white collar work, business, or the professions, and 18 per cent had done no work. "Marginal" work such as junk dealing, begging, and peddling had been done by 8.6 per cent. No information was available regarding the occupations of one-third of the sample. Likewise, no information was given on whether or not the occupations cited reflected the entire work history of the recluses or simply represented one portion of their lives (Table V).

On the whole, the group appears to have been economically comfortable judging from the amount of rent paid. For those on whom there is information, 19 per cent paid less than $60.00 per month while 4 per cent paid over $60.00 per month, and roughly 26 per cent owned their own homes. Unfortunately there was no information on almost half of the sample (Table VI).

VOL. 12 1960

Further evidence supporting the idea that this is a fairly comfortable group economically comes from the observation that nearly 25 per cent sustained themselves on their property holdings in the form of savings, dividends, and rents. Another 21 per cent did some sort of work, and only 13 per cent received outside support either in the form of welfare assistance or funds from relatives

TABLE IV. TYPES OF HOMES IN WHICH RECLUSES RESIDE

	FREQUENCY DISTRIBUTION	
	NUMBER	PER CENT
Apartment of any size	46	43.8
Own home or farm	30	28.6
Hotel room or suite	11	10.5
Shack, garage, basement, no home	7	6.7
No information	11	10.5
Total	105	100

TABLE V. OCCUPATIONAL BACKGROUNDS OF AGED RECLUSES

	FREQUENCIES	
	NUMBER	PER CENT
Manual and service work	19	18.1
White collar, business, and professional	19	18.1
Marginal—e.g., junk dealer, peddlar, beggar	9	8.6
Other—e.g., farmer, opera singer	4	3.8
Apparently none	19	18.1
No information	35	33.3
Total	105	100

TABLE VI. AMOUNT OF RENT PAID

	FREQUENCIES	
	NUMBER	PER CENT
Under $60.00 per month	23	21.9
Over $60.00 per month	4	3.8
Own home	27	25.7
Apparently no rent paid	2	1.9
No information	49	36.7
Total	105	100

or friends. However, there is no information on nearly 41 per cent of the group (Table VII).

2. Social Relationships.—Analysis of social relationships in which the recluses were involved indicates that the greater proportion had never married. Approximately 42 per cent were single, 28.6 per cent were widowed, 7.6 per cent were married, and about 3 per cent were divorced. There was no information

TABLE VII. MEANS BY WHICH RECLUSES SUPPORTED THEMSELVES

	FREQUENCIES	
	NUMBER	PER CENT
Property, e.g., savings, inheritance, dividends, rents	26	24.8
Work of any sort, including begging and junk dealing	22	21.0
Outside support, e.g., welfare, social security, unemployment insurance, neighbor's and family's support	14	13.3
No information	43	41.0
Total	105	100

TABLE VIII. MARITAL STATUS OF RECLUSES

	FREQUENCIES	
	NUMBER	PER CENT
Single	44	41.9
Widowed	30	28.6
Married	8	7.6
Divorced, separated	3	2.9
No information	20	19.0
Total	105	100

TABLE IX. TYPE OF FAMILY CONTACT OF RECLUSES

	FREQUENCIES	
	NUMBER	PER CENT
Living with a family member	33	31.4
In contact with a family member	15	14.3
No contact with a living family member	22	21.0
No living family members	20	19.0
No information	15	14.3
Total	105	100

VOL. 12 1960

TABLE X. TYPE OF LIVING ARRANGEMENTS OF RECLUSES

	FREQUENCIES	
	NUMBER	PER CENT
Living alone	59	56.2
Living with either a friend or a relative	39	37.1
No home	2	1.9
No information	5	4.8
Total	105	100

L.
2
60

TABLE XI. LENGTH OF RESIDENCE IN SAME COMMUNITY

	FREQUENCIES	
	NUMBER	PER CENT
Under 5 years	5	4.8
6–15 years	12	11.4
More than 16 years	19	18.1
No information	69	65.7
Total	105	100

on 19 per cent of the group. It may well be that the process of becoming a recluse starts at the point in life where a decision regarding marriage is to be made (Table VIII).

A surprising proportion of recluses were living with a family member who was usually a sibling. Those living with a family member numbered 31.4 per cent and another 14.3 per cent kept in touch with their families. On the other hand, an equally large proportion were isolated from family contacts; 19 per cent were known not to have a family relationship and another 21 per cent had not kept in touch with their families (Table IX).

Needless to say, most of the recluses, 56 per cent, lived alone. However, 37 per cent shared their living quarters with either relatives or companions. Apparently we must recognize a subtype which may tentatively be called "associated or joint reclusiveness" (Table X).

Most of the recluses were static though unintegrated members of their communities. The largest proportion for which there is information had been living in one community for over 16 years and another 11 per cent had been in the same community for from 6 to 15 years. Only 5 per cent lived in one community for under 5 years. Unfortunately this is another area where information is scanty, no data being available on two-thirds of the group (Table XI).

How do these communities "feel" about having recluses in their midst? Despite the large group which had lived in one community for many years, the

TABLE XII. TYPE OF RELATIONSHIP OF COMMUNITY AND RECLUSE

	FREQUENCIES	
	NUMBER	PER CENT
Community knows nothing about recluse	16	15.2
Positive: e.g., community is friendly, tolerant, helpful, etc.	16	15.2
Negative: e.g., community is hostile or rejecting. Recluse has brushes with law, welfare, hospitals, or is "expelled" to poorhouse, etc.	11	10.5
No information	62	59.0
Total	105	100

community did not know of the existence of 15 per cent of the group. In another 15 per cent of the cases, the communities appeared to feel positively toward the recluses for they either gave them concrete assistance or expressed friendly feelings toward them. However, in about 11 per cent of the cases the communities responded negatively toward them. There was no information about 50 per cent of the cases. The attitudes of the community probably depended upon how much the recluses' behavior conflicted with the existing norms. Such conflicts often resulted in invoking the help of the police as well as of health and welfare agencies in an attempt to protect either the individual recluse or the members of the community (Table XII).

3. Patterns of Behavior.—As for the predominant modes of behavior for which the recluses came to be known to their communities, 27.6 per cent were known as "withdrawn," "mysterious," or "uncommunicative." Another 26.7 per cent were considered "frugal," "shabby," or "poverty stricken." Only 5 per cent were described as "bizarre." In one of the cases described as bizarre, the recluse was reported to have set fire to money. However, there was no information on 41 per cent (Table XIII).

Undoubtedly the condition in which the recluses kept their homes accounts partly for their having been called "poverty stricken," as well as for the hostility

TABLE XIII. PREDOMINANT BEHAVIOR PATTERN OF RECLUSE AS REPORTED BY OTHER MEMBERS OF COMMUNITY TO PRESS

	FREQUENCIES	
	NUMBER	PER CENT
"Withdrawn," "mysterious," "uncommunicative"	29	27.6
"Frugal," "shabby," "poverty-stricken"	28	26.7
Bizarre or eccentric, e.g., sets fire to money	5	4.8
No information	43	41.0
Total	105	100

exhibited toward some of them. The living quarters of 37 per cent were described in the press as being in "shambles," "piled with trash," "unkempt," or "dingy." Only 7.6 per cent lived in "neat," "elegant," or "comfortable" quarters. There was no information on 52.4 per cent of the cases (Table XIV).

The discrepancy between the apparent "poverty" of these recluses and the overwhelming sizes of their estates is the most dramatic example of their eccentricity. Of the 85 per cent of the cases on whom there was information, only 5 per cent seemed to have no money at all, and 15 per cent had less than $30,000. The other 65 per cent had estates ranging from $30,000 to $5,000,000.

TABLE XIV. CONDITION IN WHICH HOME OF RECLUSE WAS KEPT

	FREQUENCIES	
	NUMBER	PER CENT
"Shambles," "piled with trash," "unkempt," "dingy"	39	37.1
"Neat," "comfortable," "elegant"	8	7.6
Apparently no home	3	2.9
No information	55	52.4
Total	105	100

TABLE XV. VALUE OF ESTATE LEFT BY RECLUSE

	FREQUENCIES	
	NUMBER	PER CENT
Apparently no money	5	4.8
Under $30,000	16	15.2
Between $30,000 and $210,000	37	35.2
Between $210,000 and $700,000	12	11.4
Between $700,000 and $5,000,000	10	9.5
Other, e.g., "jewelry," "thousands of dollars," "real estate"	9	8.6
No information	16	15.2
Total	105	100

This finding supports the previous analysis which indicated that the group was relatively wealthy. Needless to say it also supports the popular stereotype of the miserly recluse. Reference to Table I indicates that in nearly one-third of the cases reported, large sums of money discovered at death formed the major reason for bringing the recluse to the attention of the public. The prominence of money in news stories has undoubtedly led to making them easy victims for criminal attack and also to weighting this series of "patients" (Table XV).

Another popular stereotype of the recluse which was supported and probably perpetuated by the descriptions in the press, was that of "pack rat." Forty-five

TABLE XVI. MANNER IN WHICH RECLUSES UTILIZED MONEY

	FREQUENCIES	
	NUMBER	PER CENT
Most of money kept at home*	47	44.8
Money kept in bank, or invested in stocks, property	31	29.5
Had no money	5	4.8
No information	22	21.0
Total	105	100

*Information on 6 subjects reveals that they kept money at home for reasons of "safety" or because they "didn't trust banks" or "believe in banks."

per cent of the sample stored most of its money at home. On the other hand, 30 per cent had invested it in stocks or property or else kept it in a bank. Only 6 recluses gave reasons for storing their money at home and revealed that they did so for reasons of "safety" or because they did not trust or believe in banks (Table XVI).

Finally, of the 26 per cent of the cases for whom there was information, 10.5 per cent did not make provision for the disposition of their estates. Of the 15.5 per cent who did make such provision, 10.5 per cent willed it to family members or friends and 5 per cent to various organizations. As a result of this, they often left in their wake a series of family feuds, court suits, and other forms of financial and testamentary chaos.

TABLE XVII. FINAL CONDITION OF RECLUSES

	FREQUENCIES	
	NUMBER	PER CENT
Deceased	74	70.5
Physically ill	15	14.3
Judged mentally ill	4	3.8
Apparently in good health	4	3.8
No information	8	7.6
Total	105	100

4. Final Status.—What is the final status of these recluses who have been described in newspapers? The largest number, 74, or 70.5 per cent, was deceased. Of these 74, 22 died in a violent manner, 15 died of natural causes, and 37 died of unspecified causes. Of the 22 who died in a violent manner, 9 were involved in accidents, 7 apparently starved to death, 4 were murdered, and 2 committed

suicide. As for the remaining cases who were still living, 14 per cent were found to be physically ill, 4 per cent were judged mentally ill, and 4 per cent were in good health (Table XVII).

DISCUSSION

At this stage, a tentative profile of the aged recluse appears to be the simplest way to summarize the findings and to lead into a discussion of the problem.

The masculine pronoun will be used to describe our typical recluse, although the number of men and women was almost equal. The aged recluse in this selected series was about 80 years old. He lived in New York City or another eastern city and was an apartment dweller. He was equally likely to be in a middle-class occupation (such as white collar work, business or profession), a manual laborer, or never to have worked. He was economically comfortable and even wealthy, judging from the amount of rent paid and the fact that he sustained himself on his property or savings.

The recluse appears to have started on the road to seclusion early in life. This is evident from the fact that he had never married. He was as likely to be in contact with other family members as not; however, he lived alone. He had resided in his community for a relatively long period of time and was likely to be known to his neighbors, who were frequently friendly toward him.

As for the mode of behavior for which the recluse came to be known to the community, it appears that he was as likely to be considered "withdrawn" as "poverty stricken." The reason for the latter appelation stems, no doubt, from the fact that his house was in a "shambles." This apparent poverty was contra-indicated by the reality of his great wealth which was usually discovered upon his death. The community had no way of knowing about this wealth since most of the money was stored at home. Finally, the public came to hear of the recluse upon his death, usually by virtue of some violence associated with it or by news of his hoard.

Needless to say this description is limited by the special nature of this series of cases and obviously coincides with the stereotype conveyed by the press. Data on a less newsworthy sample of recluses might well yield more accurate information and permit more definitive conclusions.

One dozen recluses of this type have been reported by Erskine in her book, Out of This World.[1] Each one of these cases represents painstaking detective work in the accumulation of facts. One of the most dramatic examples is the story of the Collyer brothers. While they coincide in general with the profile presented above, the more detailed information provided in Erskine's book gives insights not to be found in more sketchy accounts. In the hope of raising some questions for research, the Collyer case will be described in detail and discussed.

Homer and Langley Collyer lived in total seclusion from 1938 until 1947 when they were found dead. Before that they had lived in near total seclusion with their mother for almost 25 years. Their father, a well-known surgeon, had gone off to live by himself. Their family was wealthy and socially prominent.

Apparently the process of withdrawal from life was started by the mother who encouraged her two sons in this way of life. Dr. and Mrs. Collyer had been first cousins which might possibly have resulted in accentuation of hereditary mental abnormalities. On the other hand, the environment provided by these parents was peculiar to say the least. The mother had been an opera singer who supposedly sacrificed her career for that of her husband. According to gossip, she had always been "unconventional"; she and her husband were rumored to have been lovers prior to their marriage. As a result of these rumors Mrs. Collyer felt that she had to shield her sons from the world.

Homer Collyer, one of the brothers, was supposedly the brightest student in the class of 1904 at Columbia College. It was remembered that he rarely left the home, and was mainly interested in theology and higher mathematics. Langley studied to be a concert pianist but interrupted his career for what to us seem strange reasons. He told friends: "My last concert was at Carnegie Hall, Paderewski followed me. He got better notices than I. What was the point of going on?"

Their lawyer, when asked if he attributed the brothers' retreat from life to the shock of their mother's death, replied that they were "odd" before she died. When she was alive all the doors were closed and the shutters drawn. When they took their mother to the opera they used one cab driver and requested that he wait outside until they all had entered their home when they returned. This driver thought that they always seemed frightened.

Fear, of course, seems to be the logical mechanism to use in order to explain the recluse's withdrawal and it is certain that the Collyers exhibited it. Langley used to say when explaining his shabby appearance: "I have to dress this way . . . they would rob me if I didn't. We purposely make our home look as if no one lived in it. We would be murdered otherwise."

Providing he has sufficient funds a recluse need not have too much contact with the world. The Collyer brothers explained to a real estate agent that they "simplified" their lives by using neither gas nor electricity. "We've no telephone . . . and we've stopped opening our mail. You can't imagine how free we feel." What cooking they did was done on an oil stove. They subsisted for the most part on peanut butter. During the second world war they had no ration tickets and only bought nonrationed foods because they "didn't believe in rationing."

Once, in 1938, when both Homer and Langley went to call on a real estate agent, they found on their return that someone had tried to break into the house. They never left the house together again. In fact, they piled boxes from floor to ceiling so that it took half an hour to get upstairs. Langley had honeycombed the entire house with tunnels and ingenious booby traps, any one of which might have killed a person attempting to pass it. In fact, at the time of their deaths, almost 50 tons of debris were removed from the house.

Needless to say the community of Harlem, in which the Collyers lived, did not take too kindly to their retreat into caverns of junk. For years bankers and lawyers tried to contact them to discuss the sale of their valuable property and mortgages. The Collyers never opened their doors or their mail. In the 1930's a Consolidated Edison representative came to remove the meters from their basement and needed police officers and firemen to assist him. There were constant rumors of Homer's death which the police attempted for years to investigate, without success. When finally an officer entered, he found Homer looking like a mummy propped up on a cot wrapped in burlap bags. Homer said: "I am paralyzed and blind . . . my legs are doubled by rheumatism . . . I can never lie down again." Why didn't he call a doctor? Homer felt that he and Langley knew too much about doctors since their father had been one.

In 1942 firemen were investigating basements for fire hazards. They pounded on the door but Langley would not let them in nor would he permit the bank to have the litter in the backyard cleared. Thus they defied orders issued by the Health and Building Departments.

Needing medical attention and ignoring Health and Fire Department regulations, the Collyers managed to live into old age to the consternation of those who were somehow involved in their affairs. The havoc and disgust they engendered were summed up by one police officer who said: "Hermits are a nuisance. There ought to be a law against them."

PSYCHIATRIC CONSIDERATIONS

Since the outstanding common characteristic of recluses is withdrawal from the world and the avoidance of social contacts, one would tend to conclude

VOL. 12 1960

that they are persons with either latent or overt schizophrenia who have successfully avoided treatment and whose behavior has, for the most part, been such as to elude institutional care. Only further intensive psychiatric study of a group of cases can substantiate or modify this viewpoint which may serve temporarily at least, as a working hypothesis. Certainly, the tendency to recluseness seems to begin early in life based on case material available and this view is substantiated by a high percentage (42 per cent) of individuals who had never married, in the group presented in this study. Since an additional 28.6 per cent were widowed, it is possible that the loss of a spouse may allow a latent tendency to recluseness to become overt.

The whole concept of "reclusiveness" may be clarified by differentiating two principle types: "absolute reclusiveness" and "partial reclusiveness." In the first group the contacts with the outside world are reduced to a minimum; in the second group, contacts are definitely restricted but may include pleasant relationships with selected neighbors or family members. One notes also another interesting type which may be termed "joint reclusiveness" often involving two siblings. Joint reclusiveness is admirably exemplified in the Collyer case. Joint recluses might, in addition, exhibit a tendency toward either partial or absolute reclusiveness.

Since the recluses here described are in the older age groups, it is appropriate to look into the possible effects of advancing years on pre-existing mental abnormalities. The best description of the interaction of senile mental disorder with schizophrenia is still that of Bleuler.[5] He wrote: "With respect to organic brain diseases, the different forms of cerebral atrophy due to advancing age very often complicate the picture of schizophrenia; it is certainly not the rule that senile dementia occurs to any marked degree in our old schizophrenic patients. In this particular complication, too, the symptoms of both diseases add up in very distinctive fashion. Disturbances of recent memory, of orientation, and poor perception are added to the schizophrenic deterioration already present. If it is still present the energy invested in delusions and actions seems to decrease. The hallucinations do not necessarily disappear; sometimes however senile hallucinatory deliria occur. The only remarkable feature is the manifestation of affects. In some of these cases hardly any response can be obtained from the patients; they vegetate, indifferent to their surroundings. Sometimes however the organic psychosis releases some of the affects; thus the patients again become somewhat more approachable." Bleuler also points out that in some of the mixed cases, in which differentiation of the two conditions presents great difficulty, one may be dealing with a latent schizophrenia which only becomes manifest with onset of cerebral atrophy.

In emphasizing the effect of an advancing chronic brain syndrome on reclusive schizophrenia, consideration must be given to the effect of the aging process, not only on the brain but on the body as a whole. Changes associated with aging are functional disorders such as increasing fatigability, diminished muscular strength, and lessened activity of neuromuscular coordinations. Disturbance of vision and hearing also occur. In addition, there may coexist hypertensive or cardiovascular disease, as well as common diseases such as arthritis

of various types, osteoporosis, benign prostatic hypertrophy, and a heightened incidence of malignant tumors. All these disorders lessen mobility of the elderly recluse and serve to accentuate the physical reasons for the tendency to withdraw.

LINES OF FURTHER RESEARCH

One need not apologize in this type of exploratory study for raising more questions than can be readily answered. Obviously the basis for all further investigation must lie in improved case finding, utilizing all possible sources rather than the limited group described here. This will involve working with welfare agencies, both private and municipal, as well as with the Medical Examiner's office, the police department and the city morgue. One may confidently expect that there are many more impoverished old men and women, living and dying alone, who never make the newspaper headlines. Such a search for the true incidence of recluses will surely raise a train of further questions worthy of detailed study.

Further research would involve the intensive analysis of the lives of a group of recluses with reference to heredity, early environment, personal relationships, work history, and the stages leading up to a full blown reclusiveness. The differentiation of the varied degree of reclusiveness and their characterization should have a profound effect on our practical attitudes and on our approach to the peculiar problems presented.

THE QUESTION OF COMMUNITY RESPONSIBILITY

These recluses form definite threats to community health in the form of fire and explosive hazards; often by harboring large numbers of cats and dogs, they become foci of vermin. Their unpredicatable behavior because of unregulated mental disease offers an invitation to criminals who have learned that old people living alone often have treasures hidden in their mattresses. The story of the Collyers is duplicated many times over in the lives of other recluses. Sometimes these are wealthy individuals, often of influential families, who had they been poor, would probably have been institutionalized or perhaps imprisoned. Although not usually as dramatic as the Collyer brothers, these cases often present urgent and pressing problems. In whom is responsibility for them vested? On which one of a variety of agencies, both public and private, can a community rely to protect both the general welfare and the unfortunate individual?

There can be no easy or pat answer to these queries. Neighbors probably constitute the first line of assistance and defense, but to what extent does a well-intentioned neighbor become his brother's keeper? What part can a practicing physician or a public health nurse play? Do city police or state troopers identify these people in their communities or areas of protective responsibilities? How often do fire departments in prevention activities learn of these fire hazards? When is the Board of Health and its officials justified in stepping into these situations?

At this point it must be admitted that as long as recluses commit no overt act against the public interest, they are entitled to be left alone. On the other

VOL. 12 196

hand their possibilities as hazards should be recognized by neighbors, landlords and building superintendents in order that they may be reported to some properly constituted authority who will then probably be glad to turn the problem over to another equally well constituted agency.

The data and the observations presented in this paper are not intended to produce action programs but rather to stimulate thinking along many lines about a small but well defined group in every community whose characteristics and welfare have largely escaped consideration. The authors are confident that other students of the medical and social problems of the aged will be encouraged to undertake more thorough studies and to arrive at sounder conclusions, and thus aid the recluse and his neighbors.

OL.
12
960

SUMMARY

1. The characteristics of the aged recluse are described on the basis of 105 cases reported with varying degrees of completeness by newspapers in the New York City metropolitan area.

2. The limitations of this method of case finding are discussed, but enough factual material has been collected to emphasize the social amd medical implications of this extreme type of aberrant behavior and to generate a typology of reclusiveness which consists in differentiating absolute and partial, as well as joint, reclusiveness. Suggestions are made for future methods of study and the value of more accurate information on incidence, as well as more intensive study along social, medical, and psychiatric lines, has been emphasized.

3. The complex aspects of community responsibility are outlined with reference to dangers to the individuals themselves, but more importantly to the community as a whole. Many of these recluses are harmless, while others constitute potential menaces to public health in the form of fire and explosion hazards, breeding places for vermin, unregulated mental disease, in resisting cooperation with community agencies, and in offering opportunities for criminals who expect all old people living alone to have large sums of money concealed.

4. In view of the increasing numbers of old people living alone, unsupervised and unprotected, further studies should alert community health and welfare agencies to the problems posed and should help to develop policies and to delimit responsibilities.

REFERENCES

1. Erskine, H. W.: Out of This World—A Collection of Hermits and Recluses, New York, 1953, G. P. Putnam's Sons.
2. Neilson, W. A., et al. (eds.): Webster's New International Dictionary of the English Language, Springfield, Mass., 1939, G. & C. Merriam Co.
3. Forty-eight clippings were taken from the New York Times, forty from the New York Herald Tribune, eleven from both of these, one from the New York Post, two from the New York Daily News, one from the New York Sun, and two from sources unknown.
4. Twenty-nine clippings represent the years 1942 to 1949, and seventy-six clippings are taken from the years of 1950 to 1959.
5. Bleuler, E.: Dementia Praecox or the Group of the Schizophrenias, Translated by J. Zinkin, New York, 1950, International Universities Press, pp. 266-267.

SCHOOL SERVICES FOR CHILDREN WITH EPILEPSY IN URBAN AREAS

HELEN M. WALLACE, M.D., M.P.H.*
MINNEAPOLIS, MINN.

From the University of Minnesota School of Public Health

(Received for publication Nov. 29, 1959)

VOL
12
196

AMONG the various types of handicapping conditions of childhood, epilepsy represents one where with diagnostic and treatment services of high caliber excellent results can be anticipated for the great majority of patients. In spite of the fact that the convulsive disorder is controlled in approximately 80 per cent of the children when they are under the care of a good service, few broad community programs for patients with epilepsy have been developed up to the present time. These programs include early case finding and careful evaluation, diagnosis, treatment, and management from the medical, health, educational, social, psychological, and vocational viewpoints; and continuous following up and and supervision of these patients. The cost of comprehensive services for patients with epilepsy is considerably lower than that for many other types of disease in children.

Even in the absence of such comprehensive services, it is of course obvious that much can be done in any one facet of care, including that of education. During 1958, when a special survey was made of school services provided for handicapped children in the larger urban communities of our country, an opportunity arose for the study of children with epilepsy. This paper will report on the facts derived from this study concerning epileptic children.

METHOD OF STUDY

During the winter of 1958 a questionnaire was sent to the health officers and superintendents of schools of each of the 106 cities of our country with a population of 100,000 or more according to the 1950 census. The questionnaire contained items regarding the types and numbers of handicapped children cared for in the public school system; the age at admission; the kinds of school facilities provided; the presence of established criteria for special educational placement, and the agency responsible for their establishment; the method of review of

*Professor of Maternal and Child Health.

applications for special educational placement, personnel engaged in such review, frequency of review; organization of special education within the public school system; method of financing and costs of education of handicapped children; and the provision of transportation and attendant service during transportation of handicapped children to and from school.

This paper will summarize the information provided on the questionnaires in regard to children with epilepsy.

STUDY FINDINGS

According to the 1950 census, the 106 cities had a combined total population of 44,311,716 or 29.4 per cent of the total population of our country, and a combined school enrollment of children aged 5 through 17 years of 7,192,100 or 28.4 per cent of the total population within these ages. Ninety-eight (92 per cent) of the 106 cities responded. These 98 respondent cities had a combined total population of 41,686,921 (94 per cent) and a combined school enrollment of children aged 5 through 17 years of 6,840,105 (95 per cent).*

OL.
12
960

TABLE I. AGE AT ADMISSION OF CHILDREN WITH EPILEPSY TO SCHOOL

AGE AT ADMISSION (YEARS)	NO. OF COMMUNITIES
7	1
6	26
5¾	5
5⅔	1
5½	1
5	25
4¾	2
4½	5
4	1
Age not stated	9
None cared for	22 (22.4%)
Total	98

AGE AT ADMISSION TO SCHOOL

All of the 98 school systems except 22 (22.4 per cent) have some provision for children who have epilepsy.

It is becoming more accepted that certain types of handicapped children may benefit from school attendance during the preschool period. Therefore, the age at admission to school has considerable importance. Eight of the school systems admit children with epilepsy under the age of 5 years. Thirty-three communities delay the school admission of children with epilepsy beyond the age of 5 years (Table I).

*The cooperation of the Health Departments and Boards of Education in providing the information is gratefully acknowledged.

TYPE OF EDUCATIONAL PLACEMENT PROVIDED

The type of educational placement provided and, of course, the quality of the service within the individual placement constitute important aspects of the care of handicapped children. To state it simply, the more services and the more comprehensive the services provided in an ambulatory school program (either special class or special day school), the less will be the need for a special residential school, home instruction, or instruction in a hospital or convalescent home.

Answers to these parts of the questionnaire were not complete. Nevertheless, it appears that at present the most frequent type of educational plan for children with epilepsy other than regular class in a regular school is home instruction (Table II).

TABLE II. TYPE OF EDUCATIONAL PLACEMENT PROVIDED FOR CHILDREN WITH EPILEPSY

TYPE	NO. OF COMMUNITIES
Home instruction only	16
Special day class only	8
Special day class, home instruction	4
Special day school only	4
Special day class, special day school	3
Special day class, special day school, hospital or convalescent home	1
Hospital or convalescent home only	1
Special day class, hospital or convalescent home	1
None cared for or not reported	60
Total	98

TABLE III. NUMBER OF CHILDREN WITH EPILEPSY REPORTED IN VARIOUS TYPES OF PLACEMENT

TYPE OF PLACEMENT	NO. OF CHILDREN
Regular class	1,373
Special day class	153
Home instruction	117
Special day school	103

A question was asked concerning the numbers of known handicapped children in the various types of educational placement. Again, this question was not fully answered by all reporting communities. From the answers provided, however, it is evident that most of the children with epilepsy are in regular class, a very desirable pattern (Table III).

THE METHOD OF EDUCATIONAL PLACEMENT OF ALL TYPES OF HANDICAPPED CHILDREN

Ninety-one of the 98 reporting public school systems (93 per cent) stated that they had criteria established for the educational placement of all types of

TABLE IV. AGENCY ESTABLISHING CRITERIA FOR EDUCATIONAL PLACEMENT OF ALL TYPES OF HANDICAPPED CHILDREN

By State Government	28 (28.5%)	
State Department of Education		27
State Department of Education and Mental Hygiene (for Mental Retardation)		1
By State and Local Government	8	
State and Local Boards of Education		8
By Local Government	46 (46.9%)	
Local Board of Education		25
Local Board of Education and Health Department		3
Miscellaneous	18	
With medical participation		7*
Without medical participation		11*
Not stated	16	
Total	98	

*Consist of one each of a wide variety of health and education personnel, including: physicians, psychologists, teachers, nurses, social workers, school counselors, and speech therapists.

handicapped children. Of the other 7 school systems, one each had established criteria for cerebral palsy, for the blind, and for mental retardation; 4 did not answer this question.

In almost half of the public school systems, responsibility for establishing the criteria for educational placement of all types has been assumed by the local government, predominantly the local Board of Education. In slightly more than one-quarter, the responsibility has been assumed by the state government, predominantly the State Department of Education. The participation of the health department, either local or state, is very small. Where the individual disciplines were listed under local government, there is medical participation in less than half of the methods (Table IV).

In half of the public school systems, the Board of Education alone reviews the applications for special educational placement of all types of handicapped children. In no instance is such review done by the Department of Health alone. In approximately one-fifth of the school systems the review is carried on jointly by the two departments, education and health. In only 3 school systems was the recommendation of the practicing physician acted upon without any agency review (Table V).

The number of professional personnel reviewing applications for educational placement of all types of handicapped children ranged from one in one school system to twelve in another school system. The most frequent number of participants was six, the number in 18 school systems. It is gratifying that in most school systems more than one professional person is responsible for the review of applications (Table VI).

TABLE V. METHOD OF REVIEW OF ALL APPLICATIONS FOR EDUCATIONAL PLACEMENT OF ALL TYPES OF HANDICAPPED CHILDREN

METHOD OF REVIEW	NO.
A (separately by Board of Education)	49
B (separately by Department of Health)	0
C (jointly by Education and Health Departments)	20
D (on recommendation of practicing physician)	3
A, B, C, D	1
A and D	5
A and B	2
A and C	2
Not checked	3
E (miscellaneous*)	13
Total	98

*Consists of a wide variety of health and education personnel, in conjunction with A,B,C,D.

The type of professional person participating in review of applications is of the utmost importance. In Table VII, the most frequent types are the psychologist and the school administrator. The nurse and the teacher participate in about one-half of the school systems, the social worker and the school counselor in about one-third. There is limited participation by such personnel as the director of special education and the vocational counselor.

In all school systems, except 14, there was some type of medical participation in review of applications. It is surprising to find infrequent participation by certain medical specialists; e.g., neurologist, pediatrician, ophthalmologist, otologist, orthopedist, and cardiologist (Table VII).

It is considered essential that there be a careful review of all handicapped children, not only prior to educational placement, but also periodically during such placement and prior to withdrawal from such placement. The questionnaire asked for information on all three aspects. Ninety-six per cent of the school sys-

TABLE VI. NUMBER OF PARTICIPANTS IN REVIEW OF APPLICATIONS FOR ALL TYPES OF HANDICAPPED CHILDREN

NO. OF PARTICIPANTS	NO. OF SCHOOL SYSTEMS
12	1
10	3
9	3
8	12
7	15
6	18
5	15
4	16
3	9
2	5
1	1
Total	98

VOL. 12 196

tems indicated that they review all applications for all types of handicapped children prior to placement, 85 per cent do so periodically during placement, and 70 per cent prior to withdrawal from placement (Table VIII). Inquiry was also made regarding the frequency of periodic review of the children during placement. Forty-five per cent of the school systems review children in special day classes at least once a year, 39 per cent review children on home instruction at least once a year, 32 per cent review children in regular classes at least once a year, 31 per cent review children in special day school at least once a year, 17 per cent review children in hospitals and convalescent homes at least once a

TABLE VII. TYPE OF PERSONNEL PARTICIPATING IN REVIEW OF APPLICATIONS

TYPE	NO. OF SCHOOLS
Psychologist	87
Administrator	87
Nurse	57
Teacher	53
Social worker	34
School counselor	33
Director of special education	16
Vocational counselor	8
Medical participants:	
Physician, type not specified	33
Orthopedist	19
Otologist	17
Family physician	16
Ophthalmologist	16
School physician	12
Pediatrician	13
Psychiatrist	9
Various medical specialists	8
Neurologist	3
Medical director	4
Cardiologist	3
Miscellaneous	4*
Miscellaneous	4†

*Composed one each of physician in Board of Education Clinics; plastic surgeon; health department; epilepsy.

†Composed one each of Assistant Superintendent of Schools; speech therapist; Consultant in Guidance; Director of Health Service.

year, and 9 per cent review children in special residential schools at least once a year. The stated range of frequency of review varied from once a week (in one special residential school and on home instruction) to a complete lack of review for children on home instruction in 2 school systems (Table IX).

Because it is recognized that "paper review" of children may not provide as complete a picture of the child, his progress, and his needs, as when the child and his family are personally seen, the questionnaire contained an inquiry regarding the provision of a team of personnel, either from the Board of Education or the Department of Health, who personally see and evaluate all children with epilepsy. Twenty-eight per cent of the communities do provide such a team, and

TABLE VIII. PLAN FOR REVIEW OF APPLICATIONS FOR EDUCATIONAL PLACEMENT, DURING PLACEMENT, AND PRIOR TO WITHDRAWAL FROM PLACEMENT FOR ALL TYPES OF HANDICAPPED CHILDREN

Prior to Placement.—	
Yes	94(96%)
No	2
Yes for convalescent patients and homebound	1
No answer	1
Total	98
Periodically During Placement.—	
Yes	83(85%)
No	5
As needed	9
No answer	1
Total	98
Prior to Withdrawal From Placement.—	
Yes	68(70%)
No	8
No answer	22
Total	98

VOL. 12 1960

the remaining 72 per cent either do not, or did not answer this question. The numbers of personnel employed on a team basis for such an evaluation vary from one person in 11 of the communities to six persons in one community. Most of the communities which do provide a team provide two or three people on the team (Table X). A psychologist and a physician (whose type was not stated) were most likely to be used. The other person used with some degree of frequency was the teacher. It is surprising to find that community facilites were rarely used, and that personnel such as nurses, social workers, school counselors, school physicians, and pediatricians were rarely used, and that neurologists were never used. Another surprising finding was that a Crippled Children's Clinic or a hospital epilepsy clinic was very infrequently used (Table XI). In 3 communities, children with epilepsy were seen for a team evaluation without a physician serving as a member of the team.

FINANCIAL ASPECTS OF EDUCATION OF HANDICAPPED CHILDREN

The source of financing the education of handicapped children is predominantly a combination of local and state tax funds (in 86 of the 98 communities). A tremendous variation in the proportions of local-state participation in financing exists, the most frequent method being equal sharing of costs by local and state governments. The most frequent method of allocation of funds used for the education of handicapped children was on a per pupil basis; a per teacher, or a per class basis is used less frequently (Table XII).

TRANSPORTATION TO AND FROM SCHOOL

Transportation to and from school is provided for children with epilepsy in 26 communities (26.5 per cent). Attendant services during such transportation is provided for children with epilepsy in 8 communities (8.2 per cent).

TABLE IX. PLAN FOR PERIODIC REVIEW OF ALL TYPES OF HANDICAPPED CHILDREN DURING PLACEMENT

FREQUENCY	REGULAR CLASS	SPECIAL DAY CLASS	SPECIAL DAY SCHOOL	SPECIAL RESIDENTIAL SCHOOL	HOME INSTRUCTION	HOSPITAL OR CONVALESCENT HOME
Once a week				1	1	
Once a month				1	2	
Once every 6 weeks					1	
Once every 2 months		1				
Once every 3 months		1			1	1
Once every 4 months			1			
Twice a year	6	9	9		8	6
Once a year	25	33	20	7	25	10
Once every 2 years	2	7	1			1
Once every 2-3 years		2	1		1	
Once every 3 years	1	2	2			
As recommended by local physician					3	6
Left to staff of institution						2
No definite plan	18	18	11	9	14	15
Frequency not stated	1	2			3	
Prior to return to school					1	
Not done at all					2	
No information or not cared for	45	23	53	80	36	57
Total	98	98	98	98	98	98

TABLE X. NUMBERS OF PERSONNEL PROVIDED FOR PERSONAL EVALUATION OF CHILDREN WITH EPILEPSY

NO. OF PERSONNEL	NO. OF COMMUNITIES
One	11
Two	8
Three	4
Four	0
Five	1
Six	1
"Team"	1
Clinic	1
Total replying	27
Total providing more than one person	16

TABLE XI. TYPE OF PERSONNEL ENGAGED IN TEAM REVIEW OF CHILDREN WITH EPILEPSY

TYPE OF PERSONNEL	NO. OF COMMUNITIES
nonmedical.—	
Psychologist	11
Teacher	5
Nurse	3
Administration	3
Director of Special Education	3
Counselor	2
Social worker	1
Therapist	1
Medical.—	
Type not specified	17
School physician	3
Pediatrician	1
Community Facility Used.—	
Diagnostic team	1
Crippled Childrens' Clinic	1

TABLE XII. METHOD OF ALLOCATION OF FUNDS FOR FINANCING OF HANDICAPPED CHILDREN

METHOD	NO. OF SCHOOL SYSTEMS
1 (per pupil)	32
2 (per teacher)	12
3 (per class)	15
1 and 2	6
1 and 3	7
2 and 3	4
1, 2, and 3	4
Per average daily attendance	1
2 and average daily attendance	1
No information	16
Total	98

DISCUSSION

These facts may be summarized as follows: (1) Twenty-two urban school services reported that they do not include children with epilepsy. (2) Thirty-three delay school admission of children with epilepsy beyond the usual age or school admission of "normal" children. (3) Most school children with epilepsy are in regular classes. (4) The most frequent type of school placement for children with epilepsy, other than regular class in regular school, is home instruction. (5) Most school systems have criteria for the special educational placement of all types of handicapped children, which have been established by state or local departments of education. (6) There has been little participation by state or local health departments, either in establishment of criteria for special educational placement or in review of childrens' applications for such placement. (7)

VOL. 12 1960

Most applications for special educational placement are reviewed by a team, most frequently composed of a psychologist and school administrator; the neurologist and the pediatrician participate very infrequently in such team review. (8) Most applications for special educational placement are reviewed prior to placement, less frequently during placement, and least frequently prior to withdrawal from placement. (9) Only 28 per cent of the communities provide a team for personal evaluation of children with epilepsy, with usually one to three people on the team; the usual team members were a psychologist and a physician (where type was not specified); a neurologist was never included in the team and a pediatrician rarely so. (10) Twenty-seven per cent of the communities provide transportation of children with epilepsy to and from school, and 8 per cent provide attendant service during transportation.

It is gratifying that most communities have established criteria for special educational placement of handicapped children. In the case of children with epilepsy, it would be desirable that such criteria be established by a team composed of a teacher from the field of special education, a neurologist and/or a pediatrician, the school physician, a social worker, a public health nurse, a psychologist, and a vocational counselor. It would be desirable also that a team composed of these disciplines review all applications for all children with epilepsy prior to placement, at least once a year during placement, and prior to withdrawal of placement. Where indicated, it would be helpful for this same team personally to see and evaluate the child with epilepsy and his family.

It is gratifying that most children of school age with epilepsy are in regular classes in school. On the other hand, it is disturbing that the most frequent type of special educational placement of children with epilepsy, other than regular classes in regular school, is home instruction. Certainly attendance at school has many more advantages for the child with epilepsy than does home instruction—increased opportunity to participate in the usual childrens' activities of a community, increased socialization, and less isolation.

It would be hoped that children with epilepsy would not have their school admission delayed beyond that of "normal" children. Such delay may contribute to any difficulty and disadvantage which a child with epilepsy may already have.

It is not surprising that community diagnostic and treatment facilities are used very infrequently for participation in school placement of children with epilepsy. As more broad community programs for children with epilepsy are developed, it would be hoped a close working relationship would be fostered between the school system and the community diagnostic and treatment facility.

SUMMARY

This paper describes the results of a questionnaire sent to all 106 cities of the United States with a population of 100,000 and over, in regard to school services currently being provided for children with epilepsy. There is considerable variation in the range of services provided as a whole. Suggestions are made for the further strengthening of such services, if children with epilepsy are to have the opportunity of reaching their full potential.

Announcements

RESEARCH TRAINING IN PSYCHIATRY. The Graduate Educational Program of the State University of New York Downstate Medical Center offers a 2 year program of research training in psychiatry leading to the degree of Doctor of Medical Science. The program is open to M.D.'s who have completed 3 years of residency training in psychiatry. Candidates will also be accepted after 2 years of residency training. In such cases, the final year of residency will be taken at the Psychiatric Division of Kings County Hospital, concurrently with this program; and a total of 3 years will be required to complete the combination of the 2 year research training program and the third year of residency. The program provides the candidate an opportunity to do research, and it offers a series of courses concerned with research methodology in the field of psychiatry. A broad interdisciplinary faculty is responsible for teaching courses in the program and for supervising research of candidates. Extensive clinical and laboratory facilities are available for research projects.

Each candidate who is accepted will be granted a fellowship of $7,500 for the first postresidency year and $8,000 for the second postresidency year. Three year candidates will, in addition, receive $7,100 for the final residency year. Applications for the academic year beginning September, 1961, should be submitted before Feb. 1, 1961. Application forms or additional information about this program may be obtained by writing to: Office of Admissions, Downstate Medical Center, 450 Clarkson Ave., Brooklyn 3, N. Y.

RESEARCH FELLOWSHIP IN PULMONARY AND VASCULAR PATHOLOGY. The Baptist Memorial Hospital, Jacksonville, Florida, plans to offer a research fellowship in lung-vascular pathology to begin in the summer of 1961. There are separate laboratories for histology, bacteriology, hematology, clinical microscopy, and chemistry and a new research laboratory with animal facilities which is approved for residency training in pathologic anatomy and clinical pathology.

Graduation from an approved medical school, 1 year of approved internship, and at least 1 year of approved residency training in pathology or in internal medicine are required.

A stipend of $3,800 per year, with an additional $350 per year each for one or two dependents is awarded. No maintenance is furnished.

A program of morphologic and histochemical aspects of various types of pulmonary fibrosis, aging, and emphysematous lungs is in progress. Facilities to permit correlation of clinical and physiologic and pathologic abnormalities are available. Current endeavor includes efforts to reproduce emphysema experimentally in dogs. A laboratory with full-time research technicians provides a wide range of special stains and histochemical and cytophysical procedures. These are also utilized in other studies, especially in neoplasia.

Major attention will be given during the fellowship to supervised research in the project of aging and emphysematous changes in the lung. The details of the duties will depend in part on the previous training and interests of the fellow.

An aspirant *pathologist* will receive carefully supervised research experience in pulmonary pathology, utilizing special cell study methods and research planning. Continued general pathology residency experience (approved) is part of the program. An aspirant *internist* will receive similar experience, with basic training in pathology as applied to internal medicine. One year of this type of training should be acceptable as Board credit.

The program is under the supervision of Dr. A. E. Anderson, Jr., Attending Internist, who is the principal investigator. Dr. Alvan G. Foraker, Pathologist, has general over-all responsibility, especially for the pathology training aspects of the program.

Applications or inquiries should be addressed to: A. E. Anderson, Jr., M.D., Research laboratory, Baptist Memorial Hospital, 800 Miami Rd., Jacksonville 7, Fla.

VOL
12
196

OL.
12
960